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# GUY'S HOSPITAL REPORTS. 107130

EDITED BY  
J. H. BRYANT, M.D.,  
AND  
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The Pharmaceutical Journal, 5, Serle Street, W.C.  
The Transactions of the Hunterian Society  
The Transactions of the Obstetrical Society of London  
Transactions of the Odontological Society (care of the Hon. Sec., Odontological Society, 40, Leicester Square, W.C.)  
Transactions of the Medical Society of London, 11, Chandos Street, Cavendish Square, W.  
The Practitioner (care of Cassell & Co., Ludgate Hill)  
The Medical Review, 43, Essex Street, Strand, W.C.  
Library of Surgeon-General's Office, U.S. Army, Washington, D.C. (per Mr. B. F. Stevens, U.S. Government Despatch Agency, 4, Trafalgar Square, London, W.C.)

- The American Journal of the Medical Sciences (care of Messrs. Lee Bros. & Co., Philadelphia, U.S.A.)
- The Brooklyn Medical Journal, c/o Dr. James McF. Winfield, 1313, Bedford Avenue, New York City, U.S. America
- The Journal of Nervous and Mental Diseases (care of Dr. C. H. Brown, 25, West 45th Street, New York)
- Transactions of the College of Physicians, Philadelphia, U.S.A.
- Transactions of the New York Academy of Medicine, care of Librarian, 17, 19 and 21, West 43rd Street, New York
- The Medical News (care of Lea Brothers & Co., 111, Fifth Avenue, Con. 18th Street, New York, U.S.A.)
- Johns Hopkins Press, Baltimore, Maryland, U.S.A.
- Le Progrès Médical (care of Dr. Bourneville, Rue des Écoles 6, Paris)
- Revue de Médecine (Monsieur le Docteur Lepine, 30, Place Bellecour, Lyons)
- Annals of the Pasteur Institute (Le Bibliothécaire Institut Pasteur, Rue Dulot, Paris)
- Mémoires de la Société de Médecine et de Chirurgie de Bordeaux (care of Dr. Demons, Hôpital St. André, Bordeaux)
- Archives d'Électricité médicale (care of M. J. Bergonié, 6 bis Rue du Temple, Bordeaux)
- Le Bulletin de la Société d'Anatomie et de Physiologie de Bordeaux (care of M. le Dr. X. Arnozan, 27 bis, Pavé des Chartons, Bordeaux)
- Verhandlungen der Berliner medicinischen Gesellschaft (care of Herr B. Fränkel, Bibliothek der Berliner medicinischen Gesellschaft, Ziegelstrasse, 10, Berlin, N.)
- Centralblatt, für Chirurgie (care of Messrs. Breitkopf und Härtel, Leipzig)
- Centralblatt für klinische Medicin (care of Messrs. Breitkopf und Härtel, Leipzig)
- Centralblatt für Innere Medicin (care of Messrs. Breitkopf und Härtel, Leipzig)
- Upsala Läkareförenings Förhandlingar (per Prof. Hedenius, Bibliothèque de la Société des Médecins, Upsal, Suède)
- Clinitchesky-Journal (c/o Dr. V. Norobieff, Vosdvijenska, maison N-4-7, logement N-1, Moscow)

# A CASE OF CEREBRAL TUMOUR IN WHICH THE SKULL WAS OPENED FOUR TIMES FOR THE RELIEF OF HEADACHE AND BLINDNESS.

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By W. HALE WHITE, M.D.

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IN these Reports for 1885 (vol. xliii.), I published an account of one hundred cases of cerebral tumour which had been in Guy's Hospital, and from them it appeared that in not more than eight per cent. of all cases of cerebral tumour could we hope to be able to excise it, for either the tumour was inaccessible, its situation could not be defined during life, it was associated with serious disease of other organs, or more than one cerebral tumour was present. Other collections of cases have confirmed this estimate drawn from the experience of Guy's Hospital, and therefore the question of the excision of a cerebral tumour has rarely to be considered, but inasmuch as headache and blindness are very common accompaniments of cerebral tumour, and can be relieved by opening the skull, the study of the annexed case may not be unprofitable.

The patient's headache began in August, 1892, and her loss of sight in the following September. At first both symptoms fluctuated, but by January, 1893, the headache was very severe and constant; she could only tell light from darkness, and optic atrophy, secondary to optic neuritis, was beginning. The headache remained very severe and persistent, and the almost total

blindness continued for twenty-one months till August 31st, 1894; the discs in the meanwhile were slowly becoming atrophied. On August 31st, 1894, some bone but no dura mater was removed and the pieces of bone were replaced. She improved so rapidly that in three days the headache had quite gone, and in three weeks she could distinguish fingers held up at a distance of five feet. Rather more than three months after the operation the sight again failed, and rather less than four months after, the headache returned; therefore on January 15th, 1895, a considerable area of bone and dura mater was removed; the headache at once disappeared and the sight improved greatly; but in two months the headache returned and the sight again failed, therefore on April 23rd, 1895, more bone and dura mater were removed. Again the headache at once disappeared and the eyesight returned. But now the improvement in both respects lasted only a fortnight, therefore on May 22nd, 1895, more bone and dura mater were taken away, with the result that the headache disappeared and the eyesight improved. This time the improvement lasted longer, and although some headache returned and the improvement in sight was not permanent, for she became blind, she was quite certain that until about a year before her death in 1900 the headache was much less than before the skull was opened.

This case illustrates admirably the benefit that may follow trephining for the relief of the headache or blindness of cerebral tumour, and is unusual in the fact that the operation was performed four times. The best evidence that it was beneficial is the fact that after the first operation the patient returned to the hospital of her own accord, with the request that she might be trephined, as she had felt such relief from the first operation, and I think that anyone, seeing the agony of the headache, and remembering that no drugs had touched it in any way, would have agreed that an operation from which relief of pain could be hoped was justifiable, especially as the same operation restored her sight. This case also teaches, what I have unhappily seen in other cases, namely, that both the headache and the blindness return after removal of bone and dura mater; but it must be



remembered that it also shows that further relief may be purchased by the removal of more bone, and that this patient was distinctly of opinion that the operations made some five years of her life endurable, inasmuch as during this time the headache was less than it was before. I never heard her express any regret that she had undergone these operations. We must bear in mind that sometimes the relief is much longer lasting than in her case. Thus, Edith Mortimer was admitted under my care on May 25th, 1898, with headache, complete blindness in one eye, considerable blindness in the other eye, and optic neuritis. She was trephined, and the dura mater opened; her optic neuritis cleared up, her headache disappeared, her sight returned, and eighteen months after the operation Mr. Stamm kindly visited her at her own home, and he reported that she had kept very well, had good sight, and only suffered from occasional headaches; she was able to do her work. Then, too, we must remember that as the expectancy of cases of cerebral tumour after they come under observation is short, trephining may relieve the pain and loss of sight for all the period of life that remains.

The principal objection to this operation is that afterwards there is sometimes a very troublesome hernia cerebri, but often even when the intracranial pressure is very great the cerebral protrusion gives rise to no difficulty. Several patients of mine, suffering from cerebral tumour, have been trephined to relieve headache and blindness, but only once has the degree of hernia cerebri made me regret the operation. Another objection is that occasionally paralysis has followed the operation. The explanation is not clear; perhaps it is that the removal of the bone enables the brain—owing to the great intracranial pressure—to be jammed against the edge of the wound in the bone. This has happened in a case in which paralysis of the left arm and left side of the face followed the operation. The tumour was a large growth, causing most fearful pain; the loss of power in the arm and face hardly mattered to the patient during the short time she lived after the operation, but it did matter to her that the agony was a little less. It has been said that patients

#### 4    *A Case of Cerebral Tumour in which the Skull was Opened*

with cerebral tumour take anæsthetics badly, but of this I have no experience, the only objections to operations I have met with are the hernia cerebri and paralysis, therefore it appears to me that in every case of cerebral tumour in which the headache is very severe, or blindness threatens and drugs have failed to give relief, the patient should be told that it may be possible to relieve the headache and avert the blindness by operation, but he should also be warned that the relief may only last a few weeks and that there is a chance of a troublesome hernia cerebri or some paralysis. My experience is that patients unhesitatingly decide in favour of operation. Occasionally it is true the operation fails to relieve the headache, but in such cases, as the intracranial tension is not increased, there is but slight risk of hernia cerebri or paralysis.

If the optic atrophy is absolute, trephining will not restore the sight, but the case here recorded shows what other cases also teach, namely, that optic atrophy may appear absolute when it is not really so, for the right disc certainly did not, before the third and fourth operations, look as though relief of pressure would improve the sight in the right eye, but it did. Further, it is interesting to note that although the improvement in the sight was so great, yet the alteration in the discs after operation was so slight as to escape ophthalmoscopic examination.

Another point that interested us much was the suddenness of the benefit after operation; headache that had lasted months disappeared at once, and even as soon as she was entirely recovered from the anæsthetic the sight had much improved. This shows what an important factor pressure is in the production of headache and optic neuritis. I think that in all the cases which I have seen trephined the benefit has been as sudden as in this.

It will be noticed that the patient now under discussion suffered from considerable anæsthesia. This varied very much at different times, and we always regarded it as a hysterical symptom, added to her symptoms of organic cerebral disease, but it is particularly noteworthy that the trephining often diminished the anæsthesia.

It is, I believe, usual in these operations to open the dura mater, but in the first operation on this patient this was not done, and yet there was considerable relief. No doubt, as a rule, it is much better to open the dura mater, but this case appears to show that if in any patient there was very great intracranial pressure, it might be worth while to try the effect of not opening the dura mater, for that can always be done at a subsequent operation. The following is an account of the case.

*First admission.*—Mary R., æt. 18, was admitted into Guy's Hospital under Dr. Goodhart, on October 14th, 1892, suffering from severe headache and blindness. There was nothing of importance in her family or personal history. In August, 1892, she was frequently sick, and suffered from pains in the head. The vomiting, after lasting three weeks, ceased, but the headache continued. At the end of September her eyesight began to fail, and on admission she was almost blind; the optic discs were swollen, the edges indistinct, and the vessels partly hidden in passing over the edges. Both knee-jerks were absent. Nothing else abnormal could be detected. For the first month after admission her headache improved, the optic neuritis diminished, and the sight got better, but during the latter part of November she had attacks of headache and vomiting. When she left on November 30th, 1892, the edges of the optic discs appeared clear, her sight was much improved, but she still had some headache.

*Second admission,* January 10th, 1893.—She was readmitted under Dr. Goodhart. She said that since November 30th, 1892, she had been sick once, and that the headache and loss of sight had returned. On admission, it was found that she could distinguish light from darkness, but she could not count fingers correctly. The visual axes were not quite symmetrical, the right eye being pulled a little outwards and downwards and the left a little inwards. The left eyelid drooped a little. There was slight obscuration just beyond the disc on the right side and considerable optic neuritis at the edge of the left disc. Both discs were very pale. She remained in the hospital till March 27th, and during nearly the whole of that time the headache was very severe. It was on both sides of the head; on the left it was

chiefly at the back, on the right chiefly over the temple. There was no change in the state of the optic discs or in the eyesight. She was not sick, but she often felt sick. On several occasions she was found to have considerable areas of anæsthesia; they varied. The accompanying chart (Figs. 1 and 2) show the distribution of the anæsthesia on February 25th, the anæsthetic areas being shaded.

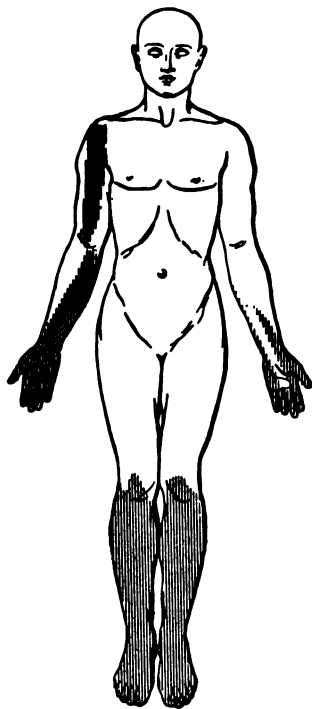


Fig. 1.

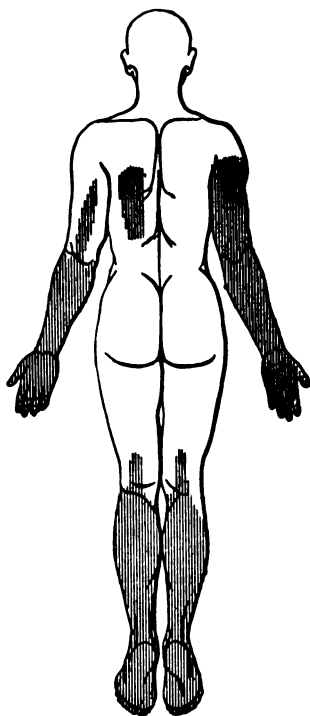


Fig. 2.

*Third admission, August 11th, 1893.*—She was readmitted under Dr. Goodhart. She stated that since her discharge on March 27th, 1893, the headache had been very severe and was now all over her head. She had been almost totally blind and since August 8th she had been very sick. The discs were now found to be very white and the vessels small and thready. She

remained in until October 8th, 1893. During the whole of that time the headache persisted very severely, her eyesight and the discs showed no change. Her anæsthesia, which included loss of sensation to touch, pain and temperature, varied. The chart (Figs. 3 and 4) below shows its distribution on admission. It is represented shaded. On September 10th the pain was very severe and in the evening she was found unconscious and unable to move, and in the morning she had no recollection of what had happened.

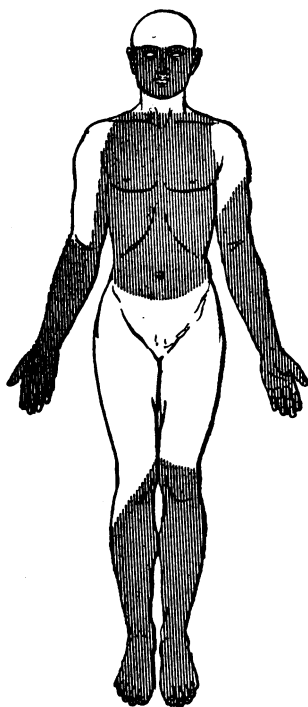


Fig. 3.

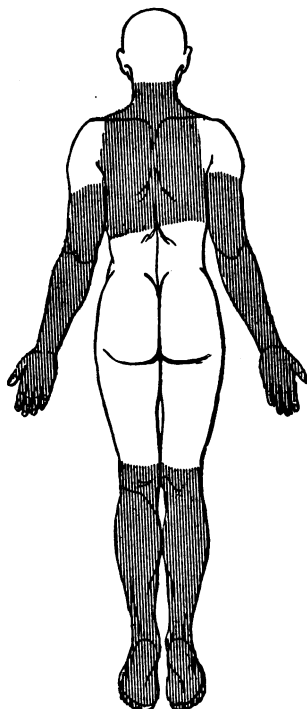


Fig. 4.

*Fourth admission, April 23rd, 1894.*—Patient was readmitted under Dr. Goodhart. She said that after her discharge on October 8th, 1893, her headache was better for three weeks, but then it returned, and it has remained very severe ever since. She

has had three attacks of vomiting irrespective of food. On admission, she complains very much of severe headache. She is weak in her arms and legs and she feels giddy if she attempts to stand. There is no oculo-motor paralysis. Both discs are extremely white, with clearly defined edges. There is some obscuration of the vessels on the top and inner side of the left disc, the right knee-jerk is very slight, the left is absent. There is a large area which is anæsthetic to pain, touch and temperature. It is shown shaded in the annexed diagrams. (Figs. 5 and 6.) The whole of the surface of the body was anæsthetic, except the hairy scalp, the ears, the margin of the eyes and mouth, the two middle fingers of the left hand and the thighs.

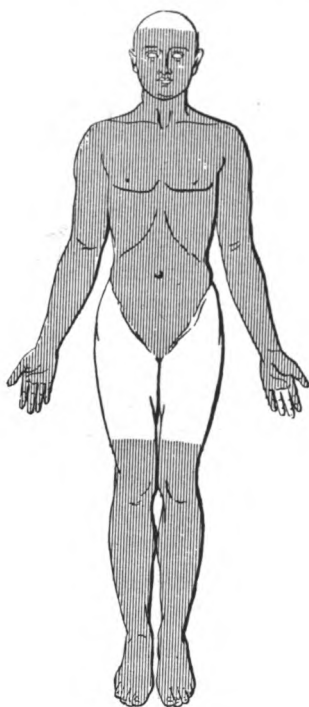


Fig. 5.

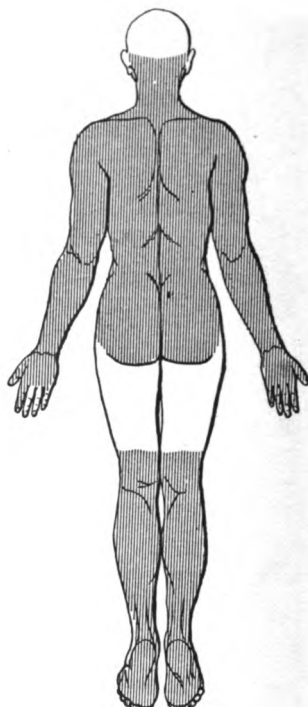


Fig. 6.

She left the hospital on May 26th, 1894, not better in any respect for her stay.

*Fifth admission, August 23rd, 1894.*—She was readmitted under Dr. Goodhart. Since her last discharge from the hospital her condition has not changed. The headache has latterly been very severe; she has been sick at intervals; she gets very tired and is often giddy; she can only tell light from darkness. The discs are the same as on the last admission, except that the obscuration at the top and inner side of the left disc has extended some distance on to the retina. Her hearing has, she says, become more acute since she has become so nearly blind. There is a tender spot over the left parietal bone a little anterior to the lambdoid suture, close to the median line. Both knee-jerks were absent. The distribution of the anæsthesia has again changed, as is shown in the annexed figures 7 and 8 over the shaded areas. She is anæsthetic to pain, touch and temperature.

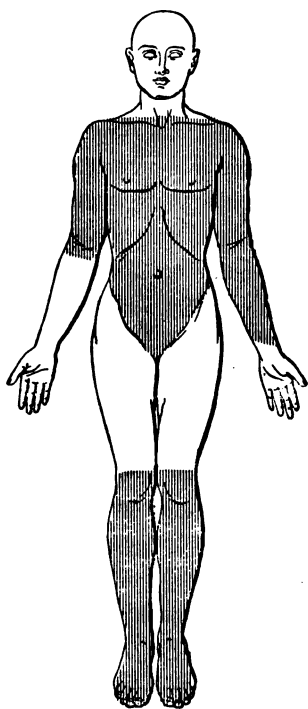


Fig. 7

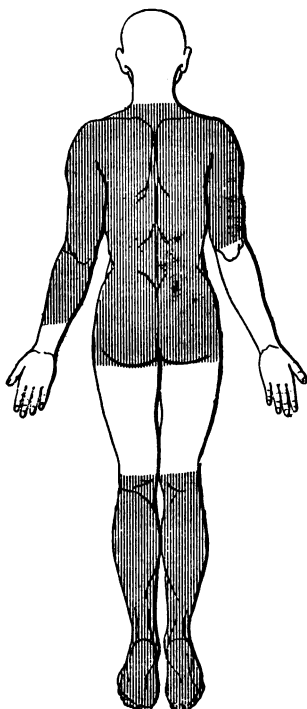


Fig. 8.

*First operation, August 31st.*—Mr. Lane removed a piece of bone about the size of half-a-crown between the left external ear and the middle line. The dura mater bulged forward in such a way as to suggest great intracranial tension. A cut was made in it sufficiently large to admit an exploring needle, which was used to see if a cyst existed in the brain; none was found, and the minute slit in the dura mater was sewn up. Pieces of the excised bone were replaced and the skin-wound was sewn up. Except for some pain during the first twenty-four hours after the operation she improved considerably, for on September 4th the headache had disappeared. She had none during her stay in the hospital which she left on September 24th, 1894. Her sight, too, improved very much, especially in the left eye, and by September 17th she could distinguish the fingers held up at a distance of five feet.

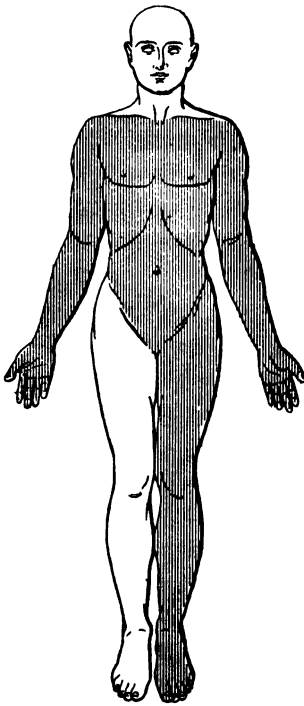


Fig. 9.

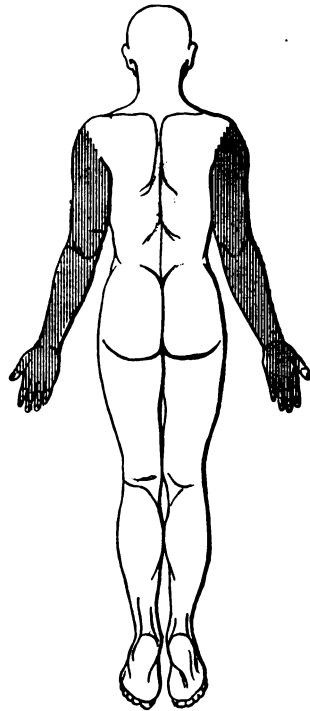


Fig. 10.



*Sixth admission*, December 20th, 1894.—She was readmitted under Dr. Hale White. She said that after she left on September 24th, 1894, she continued to improve in every respect for about five weeks, when she began to feel weaker and uncontrollable vomiting came on. Until fourteen days ago she could distinguish objects at eleven feet with the right and fourteen feet with the left eye; now she is almost blind. She has wasted considerably but her headache has not returned. The areas of anæsthesia to pain, touch, heat and cold, have quite changed (see shaded areas in Figs. 9 and 10). The arms and legs are very weak, the knee-jerks are absent. Shortly after admission the headache began again and soon became very severe.

*Second operation*, January 15th, 1895.—As the headache has been very severe since December 25th, Mr. Lane exposed the old wound; the opening in the bone was found grown over by bone, and an equilateral triangle of bone three-quarters of an inch each way was removed together with a corresponding piece of dura mater. After the operation she had some morphia, but as soon as she had recovered from this she said she had no headache, and on January 18th, she could distinguish the hand as something moving at a distance of five feet, but she sees very much better with the left than the right eye. She went out on February 10th, 1895, having had no headache since the operation and being better in every way. The condition of the optic discs had not changed. With the left eye she could count fingers at a distance of ten feet, and with the right she can see a picture on the wall at a distance of the length of the ward. There is slight deviation of the tongue to the right and slight right ptosis.

*Seventh admission*, April 18th, 1895.—She was readmitted under Dr. Hale White. She says that since her last discharge she remained free from pain until a month ago, when the headache began again; it quickly became very severe and is now continuous. Three weeks ago her sight began to grow less. The arms and legs are weak; the tongue deviates to the right. the knee-jerks are absent. The discs are in the same condition as when the last note about them was made. Her sight is as bad as before the last operation. The anæsthesia to touch,

12 *A Case of Cerebral Tumour in which the Skull was Opened*

pain and temperature occupies the shaded areas shown in the annexed diagrams. (Figs. 11 and 12).

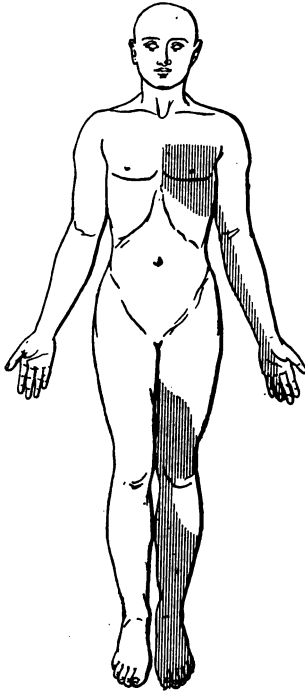


Fig. 11.

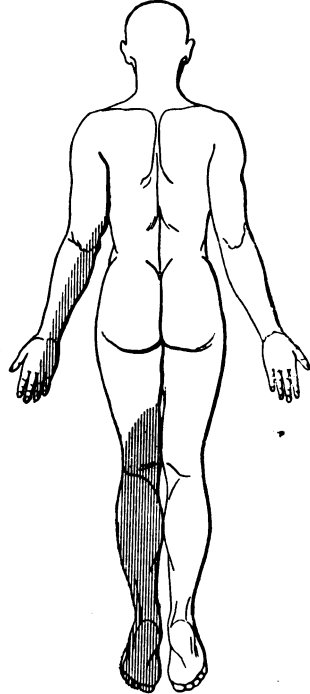


Fig. 12.

*Third operation, April 23rd, 1895.*—Mr. Lane enlarged the old opening in the bone so that it became a quadrilateral opening two inches from before backwards and one inch from above downwards. Two parallel strips of dura mater were removed, each was nearly an inch from above downwards and about half an inch across. No bone was replaced. Directly after the operation the headache disappeared and the eyesight improved considerably, and this improvement was maintained for a fortnight, but then the eyesight began to fail again and the headache returned. After this had lasted a fortnight it was decided to remove still more bone.

*Fourth operation*, May 22nd, 1895.—A piece of bone of a circumference of three inches was removed from the right side of the head, and the dura mater under it was taken away also. Directly after the operation the headache was relieved and the sight improved, so that in a day or two she could tell a bone from a wooden pen-holder. The tongue, which had previously deviated to the right, returned to the middle line. By June 11th there was some return of headache, but it was not severe. She left the hospital on June 20th, and the note states that headache was then absent and vision sufficiently acute to enable her to walk about without assistance. No anæsthesia could be detected. There was a pulsating protuberance at the site of the various operations. It was elevated about an inch.

*Eighth admission*, August 28th, 1895.—She was readmitted under Dr. Hale White, because the headache and loss of sight had returned. So much bone had been removed that it was decided not to take away any more. The right disc was unchanged, the left showed more atrophy than formerly. She left the hospital on September 23rd, 1895.

*Ninth admission*, November 12th, 1895.—She was readmitted under Dr. Hale White, on account of the headache. She was very weak; her sight was very bad again; the discs were atrophied and there was anæsthesia to pain, touch and temperature over the parts shaded in Figs. 13 and 14. She left the hospital on November 17th, 1895, not benefited in any way.

*Tenth admission*, August 14th, 1900.—The patient was readmitted under Dr. Hale White. She says that up to a year ago the headache had been but slight, and this is likely, for she had not come to the hospital. She also says that up to a year ago the headache was very much less than it was before the operation on the skull, but for the last year it has become very severe, and the vomiting is very troublesome. She is blind, except that she can distinguish light from darkness. The discs are flat, milky-white, and the vessels very small. The legs and arms are very weak; the tongue deviates a little to the right. There is anæsthesia in the whole of the right leg and right little finger; the knee jerks are absent. The headache continued very

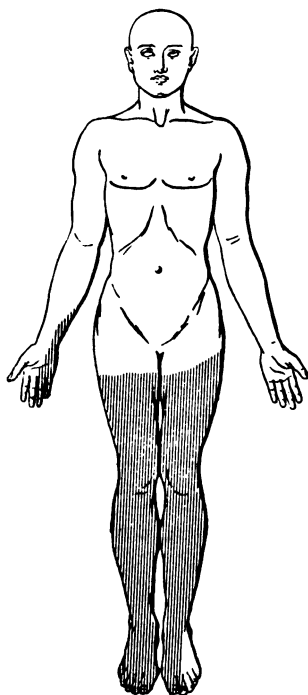


Fig. 13.

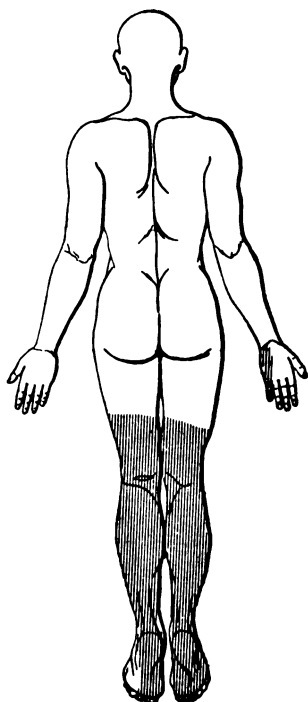


Fig. 14.

severe. On September 9th difficulty of swallowing began and she had to be fed with nutrient enemata. Soon after this signs of bronchopneumonia appeared, and she died on September 23rd, 1900, from hyperpyrexia, the temperature mounting to  $109.6^{\circ}$ .

The *post-mortem examination*, made by Dr. Bryant, took place twelve hours after death; rigor mortis well marked; hypostasis of the back and neck. There were no signs of decomposition. There was no meningitis. The brain weighed 1450 grammes: it was hardened in a 1 per cent. solution of formalin for three weeks. Four transverse horizontal sections were made (*vide* photographs). Sections 1 and 2 did not show any morbid changes. Section 3 showed the lateral ventricles to be much distended, especially their posterior cornua. The upper surface of Section 4 (*vide* photograph) showed a tumour of the left



*A Case of Cerebral Tumour, in which the skull was opened four times  
for the Relief of Headache and Blindness.*

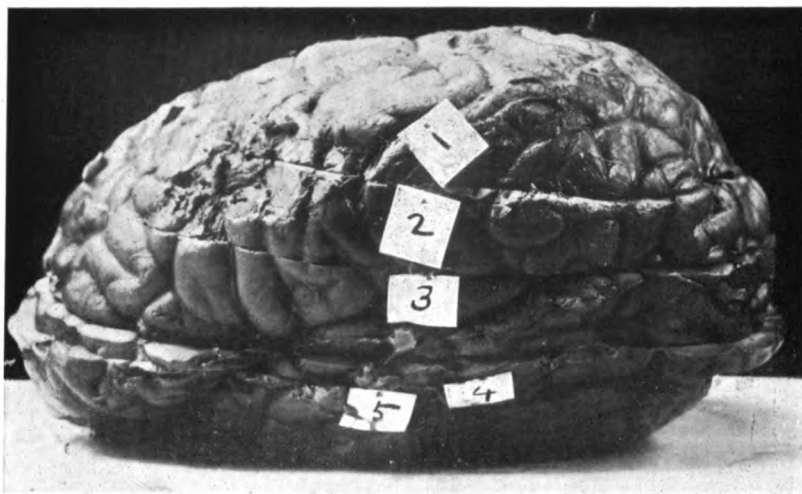


FIG. 1.—Photograph of the brain, showing the position of the sections.

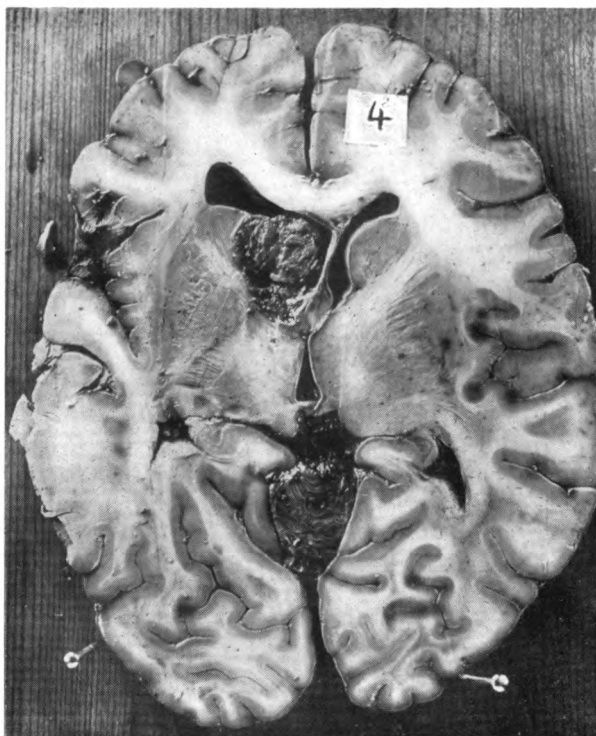


FIG. 2.—Photograph of section 4, showing the tumour involving the left caudate nucleus.

caudate nucleus which measured 20 millimetres in the transverse and 25 millimetres in the longitudinal diameter. The tumour somewhat resembled grey matter, but was of a distinctly darker shade; it bulged across the median line of the hemisphere to the right for about 5 millimetres; it did not affect the whole of the caudate nucleus, only the inner two-thirds; it did not appear to infiltrate the adjacent brain substance; it could with ease be shelled out. The spine was not examined. The cervical glands and thyroid were normal. There was no pleurisy, but numerous subpleural petechiæ. There was extensive bronchopneumonia affecting both lungs, particularly the bases. The larynx, trachea, bronchi, bronchial glands, diaphragm, mediastinum and pericardium were normal. The heart weighed 257 grammes and was normal. The aorta, pharynx, stomach, small and large intestine and the peritoneum were normal. The liver weighed 1301 grammes and was normal. The gall-bladder, pancreas, lymphatic glands and spleen were normal; the spleen weighed 96 grammes. The suprarenal capsules were normal. The kidneys were normal and weighed 246 grammes. The bladder was normal.

*Microscopical examination of the tumour.*—The growth was a spindle-celled sarcoma. It had a markedly fasciculated appearance, consisting of bundles of interlacing large spindle cells with big prominent nuclei. A few giant cells with large nuclei were seen. Several small areas of necrosis were found. In places the growth was very vascular. A few small calcareous bodies were also seen.

The sections were shown to Mr. Targett, who said he considered that the nature of the growth was quite compatible with the long duration of the symptoms.





A CASE OF  
CALCIFICATION OF THE ARTERIES AND  
OBLITERATIVE ENDARTERITIS,  
ASSOCIATED WITH HYDRONEPHROSIS,  
IN A CHILD AGED SIX MONTHS.

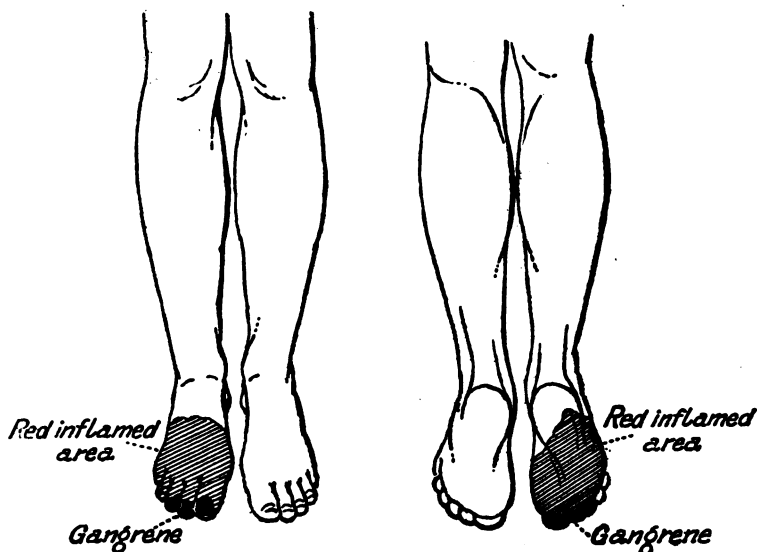
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BY J. H. BRYANT, M.D., AND W. HALE WHITE, M.D.

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E. S., æt. 6 months, was admitted under the care of Dr. Hale White, on May 11th, 1899, for general debility and wasting. He was born and had always lived at Camberwell. He was fed from the breast for the first two months, and after this, on account of his mother suddenly ceasing to lactate, was brought up on cow's milk and Robb's food biscuits. For the week prior to admission he had been given Nestlé's food. He was the second child in fourteen months. The first child was premature and still-born. The labour was difficult, it being necessary to administer chloroform. He was described by the mother as being "a beautiful baby and big enough for two." For the last three months he had lost weight and had grown very feeble. He would lie for hours without making a noise, and seemed to be too weak to cry. He was very constipated, but was never sick. No further history of any illness could be obtained.

*Condition on admission.*—Pulse 96; Temperature 97·6°. He was a miserable emaciated looking baby, lying very still and not resisting examination. There was no hair, but the scalp was covered with a scaly scurf. The head appeared to be very flat behind. The chest was not rachitic, a few rhonchi could be heard at the bases, otherwise the chest was normal. The cardiac impulse could not be seen nor felt. The heart-sounds were rapid; there were no adventitious sounds. On May 15th he vomited. On May 20th he had diarrhoea and sickness and was very feeble. He weighed 3·325 kilos. (8 lbs. 4 ozs.); May 27th he weighed 3·2 kilos. (8 lbs.). June 3rd.—During the previous week he did not suffer from much diarrhoea or vomiting. June 6th he was more feeble, and still suffered from sickness. His right foot was very cold and was rapidly becoming black. On the 8th, 10th, 12th and 13th the report states he was about the same. On the 19th he refused food. The foot was very hard and inflamed. On the 20th the foot

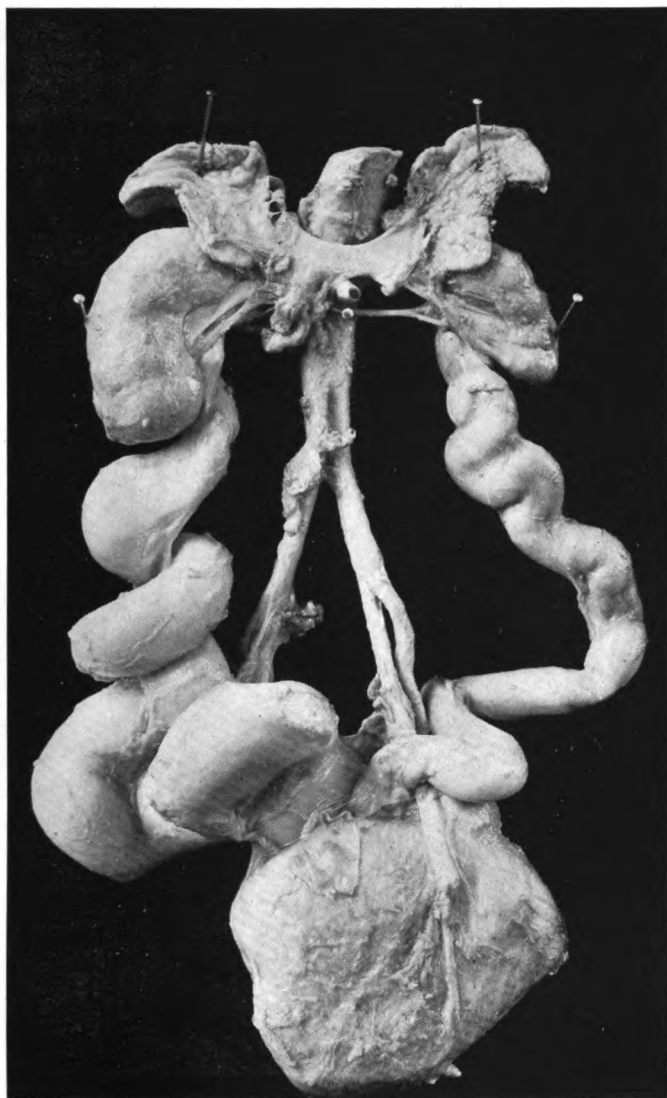


was black and gangrenous in appearance. He was very much worse, and died at 4.30 p.m. on the 21st.

The necropsy was performed on June 21st, 1899, ten hours after death. There were no signs of decomposition. Rigor mortis was well marked. The body was very emaciated. There were no external signs of congenital syphilis. The toes were becoming gangrenous (*vide* diagram). The skin was shrivelled and had a dead black appearance. On the sole and dorsum of the right foot a red and inflamed looking area of skin was seen (*vide* diagram). The femoral artery could be felt as a thick and firm cord. The brain was healthy in appearance and all the cerebral vessels appeared quite normal. The cervical glands were not enlarged. The thyroid was healthy; the thyroid arteries were rigid, thickened, hard and nodular, and their lumina were much narrowed. There was no pleurisy. Several very small nodules could be felt in both lungs. On section they proved to be tubercles arranged in small groups of five or six. The larynx, trachea and bronchi were healthy. The bronchial glands were enlarged and caseous from recent tuberculous changes. There was no pericarditis. There was no fat on the surface of the heart. The coronary arteries were thickened, tortuous and hard, and their lumina were much narrowed. The right ventricle was slightly thicker than normal; there was no dilatation. The valves and endocardium were normal. The left ventricle and auricle were normal in appearance; they were not dilated, and the myocardium appeared to be normal. The endocardium of the left auricle at, and just above the attachment of the mitral valve was hard, thickened, nodular and gritty. This was particularly well marked on the posterior and outer parts of the auricle. The gritty nodules were very small and were arranged in lines in the long vertical axis of the auricle. A similar condition was found in the endocardium lining the left ventricle, especially in the lower part over the musculi papillares. The pulmonary arteries were a little thickened and a few small yellowish patches were found in the intima of some of the medium sized branches. The intima of the aorta just above its origin up to about 1 to 2 millimetres above the level of the free edges of the valves was rough and irregular and had a feeling of grittiness. The remainder of the aorta had a normal appearance and feeling,

with the exception of the last two centimetres, which felt thickened. The intima presented a large number of small light greenish yellow spots and patches which also felt hard and gritty. The iliac arteries were very much thickened and felt firm, hard and rigid, like pipe stems. The lumina were much narrowed, in fact almost obliterated. The external and internal iliac arteries on both sides were similarly affected, but the vessels on the right more than those on the left. The intima presented a yellowish, shiny, glazed, or rather varnished appearance; it felt hard and brittle, and when bent cracked with ease. A transverse section showed much narrowing of the lumen which gave the impression that it had been caused by a thickening of the tunica media. On the whole, the appearance resembled atheroma rather than primary calcification. Both the femoral arteries and their branches were affected in a similar manner, the right, however, more so than the left. The small arteries of the lower part of the legs and the feet were almost obliterated, especially the right *anterior dorsal* and the right external plantar. The brachial artery and its branches, the thyroid, the mesenteric, hepatic and renal arteries were all thickened and rigid and showed changes similar to those described above. The stomach and the intestine had a normal appearance. There was no peritonitis and no ascites. The liver appeared to be normal, except for the hepatic arteries, which were thickened, hard, rigid, somewhat nodular and narrowed. The gall-bladder, pancreas and lymphatic glands were healthy. The spleen contained a number of small tubercles; the splenic arteries were affected in the same way as the arteries already described. The suprarenal arteries were also affected. The left kidney measured 3 centimetres in length; it felt as if it was hollowed out; its pelvis was much dilated. The right kidney measured 4·2 centimetres in length; it was much firmer than the left, but it also felt as if it was hollowed out, and its pelvis was also dilated; in fact both were in a condition of hydronephrosis, although not enlarged. Both ureters were very much dilated and were extremely tortuous; the right was much more dilated than the left and was as large as a piece of the small intestine, in fact, after the intestines had been removed in the usual manner by the

*A Case of Calcification of the Arteries and Obliterative Endarteritis,  
associated with Hydronephrosis, in a child aged six months.*



Photograph showing hypertrophied bladder, dilated ureters, and calcified arteries.



post-mortem room attendant, it was thought, on looking at the peritoneal cavity, that he had left some of the intestine behind. In its most dilated part the right ureter measured 6·5 centimetres in circumference, the left 4 centimetres. The bladder was much hypertrophied, and was also dilated; just above the neck the wall measured 5 millimetres in thickness. Neither of the ureters were obstructed. The urethra itself was not dilated. There was a very marked degree of phimosis, the urine having to pass through an orifice no larger than a pin-prick.

*Microscopical Examination.*—Sections of the right anterior tibial artery, and of the kidney were prepared by Dr. Stevens, and we are also indebted to him for the drawings of the microscopical appearances of these structures. The specimens were stained with eosin and hæmatoxylin.

The anterior tibial artery was selected as it was markedly affected. When examined with the low-power (Zeiss A, eye-piece, 1), the following main features could be made out.

1. The lumen was much narrowed and was almost entirely occluded by an organising ante-mortem thrombus.

2. The intima was enormously thickened, being quite five or six times wider than normal.

3. The elastic lamina was very distinct, it was intact, and was not so crinkled in appearance as in a normal artery.

4. There was well marked calcification, especially of the tunica media.

5. There was loosening of the tunica adventitia.

6. There was thickening of the vasa vasorum.

Examined under a higher power (Zeiss D) the lumen of the vessel was seen to be much narrowed, and it was almost filled with an organising ante-mortem thrombus. In two places (*vide* fig. 3), the epithelial layer of the intima was separated from the adjacent subendothelial layer by a portion of the organising thrombus, otherwise the innermost endothelial layer appeared to be normal. The main thickening of the artery was due to a marked proliferation and increase in size of the subendothelial layer of the intima, the tissue forming which presented a markedly

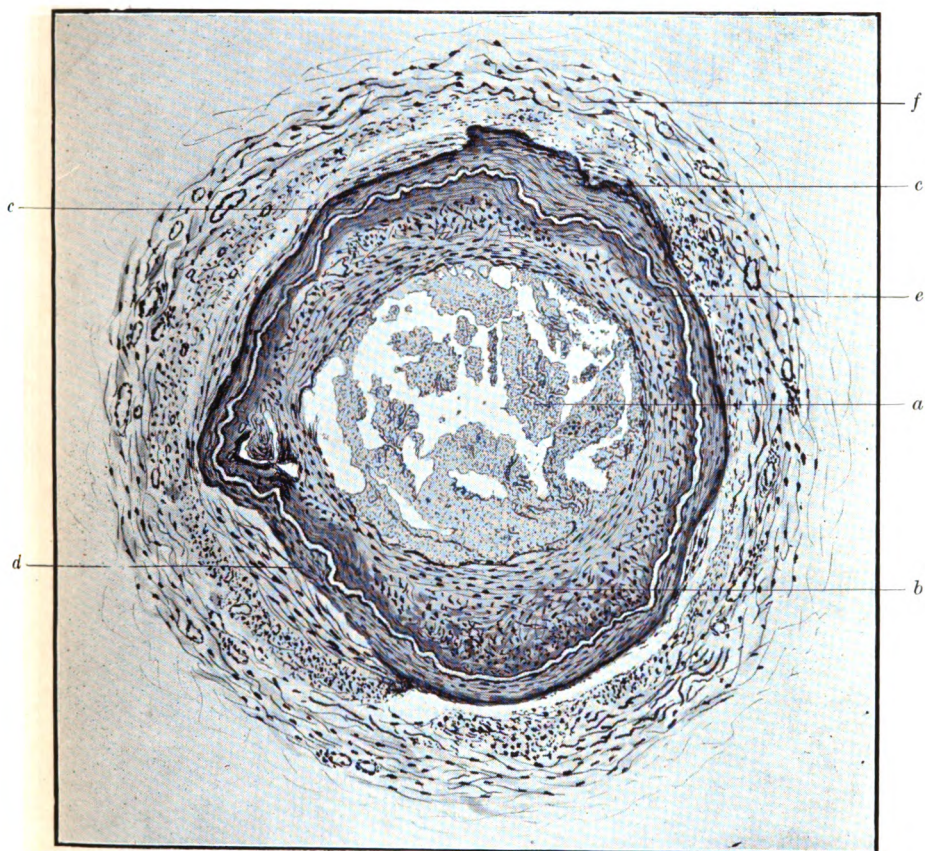
degenerated appearance, it being very granular-looking, and containing few nuclei. Immediately adjacent to the elastic layer or fenestrated membrane of Henle, there was a continuous ring of calcareous degeneration, which had stained a deep bluish violet with hæmatoxylin; it presented a somewhat hyaline and granular appearance. It varied very considerably in thickness, in places it was thin, whereas in others it occupied nearly one-half of the thickness of the subendothelial layer. Between the markedly calcified outer zone of the subendothelial layer and the degenerated inner portion, several small, rather dark brownish-looking spots and areas of what appeared to be granular debris were seen. The elastic layer or fenestrated membrane of Henle showed up as a bright, clear, continuous yellow line. It was intact, and was much less crinkled than usual. The tunica media showed extensive degenerative changes and calcification, quite the inner five-sixths of it being involved, and it was stained a deep bluish violet, with the hæmatoxylin just as the outer part of the subendothelial layer of the intima. It also presented a somewhat hyaline and granular appearance. Externally, it was bounded by a thin, more deeply-stained zone. In some parts of the artery it was bounded by a thin layer of involuntary muscular fibres, with long, well-stained nuclei, varying in thickness from one or two, to ten or twelve muscle fibres lying parallel to each other. This condition is well represented in the drawing. In the inner degenerated zone only a few small, scattered, faintly-stained nuclei could be seen, nearly the whole of this part having undergone extensive calcareous degeneration, and nowhere in this degenerated part could the outline of any individual muscular fibres be made out.

The tunica adventitia did not show any definite morbid changes. There did not appear to have been any periarteritis. There was no cellular infiltration. The connective tissue seemed to be loosely arranged; but there was no evidence of any marked degenerative changes. The vasa vasorum were a little thickened; they were not thrombosed.

*The Kidneys.*—The fibrous capsules were considerably thickened. The most marked change was the general dilatation of the tubules.



*A Case of Calcification of the Arteries and Obliterative Endarteritis,  
associated with Hydronephrosis, in a child aged six months.*

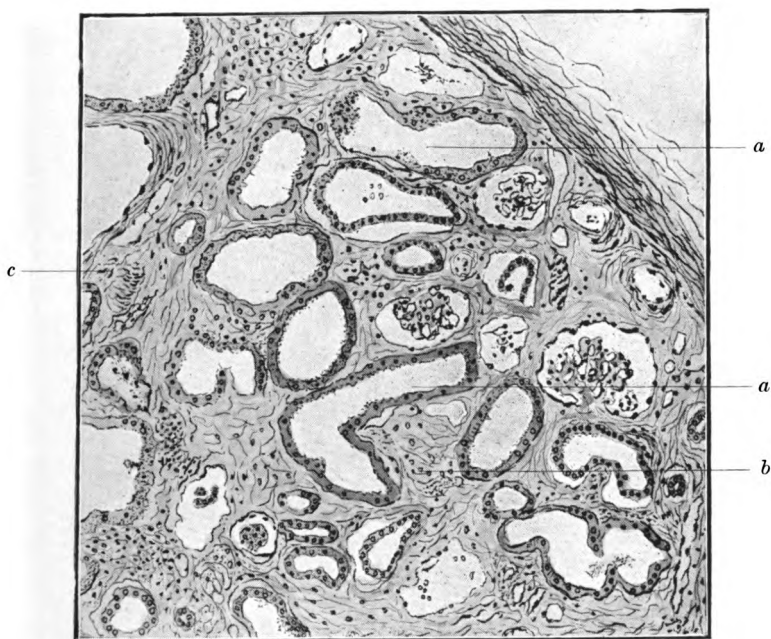


THOS. G. STEVENS (del.)

- a.* Ante-mortem thrombus.
- b.* Thickened tunica intima.
- c.* Calcified tunica intima and media.
- d.* Muscle of tunica media.
- e.* Fenestrated membrane of Henle.
- f.* Tunica adventitia.



*A Case of Calcification of the Arteries and Obliterative Endarteritis,  
associated with Hydronephrosis, in a child aged six months.*



THOS. G. STEVENS (del.)

- a.* Dilated renal tubule.
- b.* Increased growth of fibrous tissue
- c.* Thickened arteriole.



Most of them were lined by epithelium, which appeared to be stretched and flattened, the individual cells being much elongated and very little deeper than the diameter of their nuclei. In some of the tubules the cells were so flattened that the nuclei were compressed, and were altered from their normal circular to an oval shape. In a good many of the tubules the epithelium had separated from the subepithelial layer. Some of the tubes which were most dilated had no lining of epithelial cells; but contained a few epithelial cells lying loose in the centre of their dilated lumina. The epithelium of a few of the tubules had a markedly granular appearance, the nuclei of some of these cells being invisible, and very faintly stained in others. The glomerular capsules were markedly dilated. The glomeruli themselves were healthy in appearance. In places there was a very marked increase in the amount of interstitial connective tissue. Several sections were examined, and all the arteries, even to the smallest arterioles showed a remarkable amount of thickening, as much, if not more, than is usually seen in an advanced case of chronic interstitial nephritis.

*Remarks.*—There are many points of interest to be considered in this remarkable and, as far as we know, unique case. First of all there is the condition of hydronephrosis, dilated ureters and hypertrophied bladder; secondly the advanced general arterial degeneration and calcification which had led to gangrene of the toes; and lastly the ætiology of these two conditions and their relation, if any, to each other.

Hydronephrosis in children is probably almost always due to some obstruction to the passage of the urine in the ureter, bladder, urethra or prepuce, but often the precise nature of the obstruction cannot be made out. Of ten cases collected by Holt<sup>1</sup>, in only three was a sufficient degree of obstruction found. In our case both kidneys were involved, both ureters were markedly dilated, and the bladder was much hypertrophied, so that the obstruction must have been in the urethra or prepuce. The urethra was not dilated. The prepuce, however, was very tight, and could not be drawn over the glans penis for any distance. It required a very

<sup>1</sup> Holt. *Diseases of Infancy and Childhood*, p. 608.

careful examination to find the preputial opening, and it was only discovered when an attempt was made to draw the prepuce over the glans. It was a minute opening, about the size of a pin-prick, and this tiny orifice was the only one through which the urine could have passed. Such a condition must have caused a good deal of difficulty to the passage of urine. The prostate was not enlarged and there was no vesical calculus. So it appeared that the phimosis was the cause of the hypertrophied bladder, dilated ureters, and hydronephrosis, for it was the only obstruction found. Against this view was the fact that the urethra was not dilated, and also that phimosis is comparatively common and hydronephrosis very rare. In our case, however, the phimosis was extreme. We have already mentioned that occasionally no obstruction is detected, and we have found such a case recorded by Courvelain. (Bull. et Mem. de la Soc. Anat. de Paris, March, 1900). The bladder of the foetus was so distended and its abdomen so swollen in consequence, that puncture was necessary before delivery could be effected. Five hundred and fifty grammes of clear, lemon yellow, highly albuminous fluid was withdrawn. The child was a female and weighed 2.835 kilogrammes. The abdominal distension of the child was found to have been due to retention of urine. The ureters and the renal pelves were dilated. No malformation, calculus, or stricture was found. Similar cases are mentioned in this paper as having been recorded by Gaüdon, Cornelli, and Lefour. Porak has reported a case in which the obstruction was found to be due to a valvular fold in the mucous membrane, a condition which may possibly exist in other cases but may easily be overlooked.

In two of Holt's cases the ureters were so large as to be mistaken for coils of small intestine, as in our case. Not only do the ureters in these cases resemble a coil of small intestines, in size, but also in their appearance of thinness and semi-translucency; they are also, as a rule, much elongated, sacculated and twisted, which condition can be well seen in the photograph of our case. (Fig. 2).

In two of Holt's cases "typical examples of the atrophic form (contracted kidney) were seen." One of these children died at

the age of one month. In our case there was little kidney substance left, and a microscopical examination of it showed dilated tubules, interstitial fibrosis and thickening of the arteries such as is seen in chronic interstitial nephritis, a condition we shall have to again consider when discussing the pathology of the general arterial degeneration. One of Holt's cases was remarkable; the child died of what appeared to be marasmus. Double hydronephrosis was found, and the ureters were much dilated, measuring three fourths of an inch in diameter. The right kidney had an adherent capsule and was very nodular on the surface. In the cortex, just beneath the capsules, there were several small cysts containing pus. The left kidney was in a condition of hydronephrosis the cortex being very thin. Microscopical examination showed chronic diffuse nephritis of the atrophic variety; the capsule of the right kidney was much hypertrophied and there were several small abscesses beneath it in the cortex, the rest of this kidney was converted into dense fibrous tissue. The walls of the bladder were much hypertrophied. The urethra and prepuce were normal. No mention is made in any of these cases of degenerative changes in the arteries.

Another point of interest in our case is the consideration of the time at which the change commenced. The above mentioned cases show that the hypertrophy of the bladder, dilated ureters, and hydronephrosis may be brought about in utero, and we are of opinion that taking into consideration the very marked changes in our case that its origin must have been congenital.

We now pass on to the consideration of the pathology of the changes which were found in the arteries. These were extreme and had led to gangrene as a result of obliteration or great narrowing of their lumina, in fact, the case may be described as senile gangrene occurring in a child six months old. To briefly recapitulate these changes; there was enormous thickening and calcification, which rendered the vessels hard and rigid like pipe-stems, so that on bending them they would snap with a crack. The microscope showed enormous thickening of the intima with calcareous degeneration in this layer, and also calcareous degeneration

involving nearly the whole of the media, changes more compatible with a patient eighty years of age than a baby of six months.

In addition to this calcareous degeneration of the arteries, two other marked pathological changes were found, each of which would well account for the death of the child, viz., the hydronephrosis and atrophic fibroid condition of the kidneys, and the acute tuberculosis of the lungs and spleen. Naturally the first question which arises is—Was the arterial degeneration secondary to either of these conditions? We do not think the tuberculosis had anything to do with it. This was probably a late infection predisposed to by the emaciated condition of the child and may be looked upon as the actual cause of death. There were no old foci of tubercular disease, and it was evident that the extensive changes in the arteries were of much older date than the tuberculosis, which was obviously quite recent.

With regard to the renal condition, we do not consider that it was adequate to account for such very marked arterial changes. We have never seen, even in an adult, such marked degenerative changes as a result of advanced chronic interstitial nephritis or hydronephrosis, and we have not been able to find a single case recorded of such extensive arterial degeneration in children in association with either chronic nephritis or hydronephrosis.

The arteries really presented the changes not only of what might be expected in an advanced condition of chronic interstitial nephritis, but also the primary calcification of the tunica media, which is associated with old age.

The calcification of the endocardial lining of the left auricle and ventricle was also quite different in appearance to the patches of atheroma which are sometimes found in the endocardium covering the mitral valve in cases of advanced chronic nephritis.

That the change was something more than a primary calcification is also evident, for in the primary calcification of the medium-sized vessels, which occurs in old people, the tunica media is the part which is involved, it becoming first fatty, and then calcareous, until this coat may be represented entirely by a brittle calcareous tube. The intima and adventitia are, as a rule, very little, if at all affected. In our case there was remarkable thickening of the



tunica intima, which makes it quite clear that the morbid process was something more than a mere primary calcification.

The remarkable thickening of the tunica intima, which may be described as quite five or six times wider than a normal intima, certainly points to syphilis as a possible cause, for syphilis is certainly more likely to cause an obliterative endarteritis than any other disease, although marked thickening may occur in cases of chronic interstitial nephritis.

There certainly was no positive evidence of congenital syphilis; we examined all the other organs histologically and found no evidence, with the exception of the changes in the arteries. The liver was examined particularly, but it showed no sign whatever of intercellular cirrhosis. There is perhaps one point in the family history which is suggestive of syphilis, and that is, the mother was delivered prematurely of a still-born child fourteen months before the birth of this child.

The degree of the arterial degeneration pointed to a commencement of the disease in utero, another point which we consider to be in favour of a syphilitic origin.

Both endarteritis and atheroma are acknowledged results of syphilis, but such marked general calcification is unusual, and we are inclined to look upon the arterial changes, as being due to more than one cause. We consider that without much doubt syphilis was the main factor, but that the hydronephrosis and chronic fibroid changes in the kidneys played their part by raising the general arterial tension. The calcification we look upon as showing an extraordinary tendency of the tissues to early degeneration, but consider that it must be looked upon as a secondary condition.

Durante (Bull. de la Soc. Anat., January, 1899, and *Epitome B. M. J.*, vol. 1, 1899, No. 542) records a case of "congenital atheroma." The patient was a seven months' child, and was seen fourteen days after birth. It was suffering from general œdema, redness of the skin and periumbilical lymphangitis. Death resulted from peritonitis. At the post-mortem examination the organs were found healthy, with the exception of the peritonitis. The thymus was small and pale. There was no pericarditis nor

valvular disease. The pulmonary artery presented a hard condition of its walls, with here and there patches of considerable density, similar to the appearance usually seen in a senile aorta. These hard plaques broke when they were bent. The internal surface of the vessel was white, but was quite smooth. There were no naked-eye changes in the aorta; but it felt more rigid than normal. Microscopically the pericardium, endocardium and myocardium were found to be healthy. The tunica media of the pulmonary artery showed fatty degeneration and calcareous infiltration, the inner part of the coat was mainly affected, the muscular fibres in the outer part of the coat appearing to be fairly normal. In other parts of the vessel the middle coat showed only a little fatty degeneration, and in places the neighbouring cells of the inner coat were involved. Similar changes, but to a much less degree, were found in the aorta. In none of the vessels was there any appearance of endarteritis. He looked upon it as congenital, the lesion being too marked to have developed so soon after birth. No details could be ascertained as to the health of the parents. Probably one or both were syphilitic.

The changes recorded in the above case were not so advanced as in our case, for the only obvious lesions were in the pulmonary arteries. The tunica media was mostly involved, and it was definitely stated that there was no appearance of endarteritis in any of the vessels.

# SOME REMARKS ON TRANSFUSION AND VENESECTION.

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By A. P. BEDDARD, M.D.

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TRANSFUSION is the rapid introduction of fluid into the vascular system, and is usually performed by injecting at a low pressure one or more pints of fluid at the blood-temperature into the median-basilic or other vein. The fluid most frequently used is cooled boiled tap water, containing sodium chloride in the proportion of one drachm to the pint. Formerly the term transfusion was used to indicate the injection of blood, whole or defibrinated, as opposed to the infusion of salt solutions. The use of blood was founded on the assumption that the nutritive properties of the injected fluid were of importance; it is now known that this is insignificant as compared with the quantity of fluid injected. Since the injection of blood has many serious dangers and drawbacks it has been abandoned in favour of the injection of salt solutions, to which the term transfusion is now universally applied. In certain cases fluid may be introduced into the vascular system equally well, though less rapidly, by injecting the salt solution into the large intestine, or the loose subcutaneous tissue of the axillæ or flanks; but unless otherwise stated, the word transfusion will in this paper apply primarily to intravenous transfusion.

Before discussing the clinical indications for transfusion or venesection, it will be well to recapitulate what is known of the physiological effects of increasing or decreasing the quantity of

fluid circulating in the blood-vessels under varying conditions. The normal quantity of blood in circulation may be taken as about  $\frac{1}{14}$ th of the body weight, and in a man weighing 70 kilos would amount to 5,000 c.c.

Taking first the intravenous injection of salt solution, the effects of such transfusion will differ considerably, according as the arterial blood-pressure is previously normal or subnormal.

(1) If into a normal man from 800 to 4,000 c.c. of salt solution were injected, the arterial blood-pressure would rise a few m.m. Hg. directly the injection began, and then would remain stationary; on the other hand, the pressure in the big veins would rise continuously during the injection, until at the end of the injection it would be trebled or quadrupled. At the end of the transfusion the blood would be more watery and its specific gravity lower, and there would be a great increase of total fluid in the blood-vessels; in other words, a condition of hydræmic plethora. This enormous increase in circulating fluid would be accommodated partly by dilatation of the abdominal arterioles, but chiefly by stretching of the thin-walled abdominal veins, and hence the rise in venous pressure. The rate of the heart-beat would not be altered, but the work done by both ventricles would be at least trebled, because, owing to the rise in venous pressure, the input of blood into the heart per unit of time, and consequently its output into the aorta, in other words, the blood passing through the heart in a given time, would be trebled. The heart, in order to accommodate this increased intake of blood during diastole, would dilate, and at the end of systole would contain a corresponding increase in residual blood. This great increase in the work of both ventricles would not be accompanied by any increased rapidity in the coronary circulation, depending as it does on the difference in blood-pressure between the aorta and the right auricle; there would thus be increased muscular work without increased blood-supply to the working muscle. Should the heart be unable to perform the work under these circumstances, it would dilate still further, owing to accumulation within the ventricles of blood not pumped out during systole, this stretching of the cardiac muscle would stimulate the sensory endings of the

vagus in the heart and so reflexly call forth the cardio-inhibitory action of the vagus to its aid. The good of this action is to reduce the work of the heart by prolonging the diastole and slowing its rhythm. The intake and output per beat are thus increased but the output per minute is decreased, hence the arterial blood-pressure falls. On the other hand, blood accumulates in the veins through its not being passed on by the right ventricle, therefore the venous blood-pressure rises. A decrease in the work of the heart and a corresponding decrease in the rate of the coronary circulation are thus produced.

At the end of about an hour, however, the whole circulation would probably have returned to normal, and the hydræmic plethora have passed off. When the volume of fluid in the blood-vessels is greater than normal, water is removed from the blood by the tissues generally, but especially by the muscles, until the volume of blood is again normal. The quantity of water which the tissues can take up is limited, but under normal conditions they could remove in three hours an excess of fluid equal to the whole normal volume of blood. The drier the tissues were the more fluid they could remove from the circulation, thus they could take up more in summer, or if the man had been starved of fluid for some hours previously.

(2). If, however, the arterial blood-pressure is abnormally low, whether from severe hæmorrhage or from dilatation of the abdominal arteries and veins due to paresis of the vasomotor centre, the effects of transfusion are quite different. In both cases the condition of the vascular system will be that the venous as well as the arterial blood-pressure is below normal, hence the intake, output, and work of the heart small, the coronary circulation poor, and therefore the contractions of the ventricles quick and weak, as a result the pulse will be rapid and weak and the artery badly filled.

If a man in this condition be transfused the immediate effect is to correct all the defects in the circulation—the venous pressure rises, the intake and output of the heart are increased, the arterial blood-pressure rises rapidly and continuously, the coronary circulation is improved, and the contractions of the ventricle are

more forcible and slower. It is true that the work of the heart is increased, but the heart can suffer just as much from too little as from too much to do, and within limits of time and extent, the more work a healthy heart is given to do, the better it does it, always provided that the coronary circulation is correspondingly improved.

The permanence of these good effects, however, is quite another question. In the case of hæmorrhage it has been found experimentally that if as much salt solution be injected into a vein as blood has been lost, the arterial and venous pressures will return practically to what they were before the bleeding and the good effects are permanent. Of course the blood is watery, its specific gravity is low; in fact there is hydræmia, but the all-important thing is that the quantity of fluid in circulation has been restored to normal; new corpuscles and proteids are rapidly added later.

In the case of general vasomotor paralysis, however, the effects are much less permanent, unless the vasomotor paralysis itself passes off. Owing to the wide dilatation of the arteries and veins, the capacity of the vascular system may be doubled, hence the quantity of blood which before adequately filled it, is now not nearly sufficient to do so. As fluid is transfused it temporarily fills the arteries and raises the arterial blood-pressure, but the fluid gradually finds its way into the dilated veins, and the arterial pressure falls, but not quite to its former level. It is said that by injecting more than 2000 c.c. of salt solution in this condition the fluid no longer raises the arterial blood-pressure, but simply goes on distending the veins. Here, the total result of a most copious transfusion is to raise the arterial pressure permanently to some extent, but probably not nearly to normal as long as the vasomotor paralysis lasts, to produce considerable hydræmic plethora, and consequently to greatly increase the work of the heart.

The effects on the circulation of loss of blood vary considerably with the extent of the loss. The only kind of hæmorrhage considered here will be sudden loss of various quantities of blood. The secondary results of continued losses of small quantities at a time are outside the present subject. Further, it is necessary to state that whatever the extent of the sudden loss is, so far as

is known at present, the symptoms are due almost entirely to the loss of fluid as opposed to the loss of proteids and corpuscles.

While hæmorrhage is going on there is always a fall of blood-pressure, arterial and venous, throughout the body; but provided that the loss of blood is less than three per cent. of the body weight—between  $\frac{1}{4}$ th and  $\frac{1}{2}$ th of total blood—the arterial blood-pressure rapidly recovers itself. This is brought about by increased activity of the vasomotor centre, which by vasoconstriction increases the peripheral resistance, and also decreases the total capacity of the vascular system.

There is a second mechanism for refilling the vascular system with fluid, which is nearly as important though it acts a little less rapidly, and that is the fluid in the tissues. Directly fluid begins to be lost from the vascular system, the tissues begin to give up theirs to the blood in quantities which are appreciably large, but will vary with the quantity they happen to contain. In fact, the tissues have to be looked upon as a reservoir which can either take in or give up fluid in order to keep the volume of circulating blood constant.

When, however, the quantity of blood lost is greater than three per cent. of the body weight, a large and suddenly dangerous fall of blood-pressure is produced, which is more or less permanent, and cannot be made good by the combined actions of the vasomotor centre and tissues. The condition of the circulation in such a case has already been described.

Venesection, or the withdrawal of a few ounces of blood from a vein, has a somewhat different effect upon the circulation from removal of the same quantity of blood from an artery. During the actual withdrawal of blood from the vein the quantity of blood reaching the right ventricle, that is the intake of the heart, is decreased, therefore at the end of diastole the ventricle will not have to hold as much blood as it did before, and will contract up till the blood it receives just fills it. The full ventricle will now contract and send out exactly the same quantity of blood as it did before, *i.e.*, the output of the heart, and consequently the arterial blood-pressure is unaltered. By venesection, then, the intake of the right ventricle is decreased without change in the

output of the heart. Consequently both ventricles at the end of systole will contain less blood and be smaller than before; in other words, the quantity of residual blood has been reduced. It is only necessary now to show how and under what circumstances this will help the heart. Changes in the size of the ventricle during diastole will affect, in two opposite directions, the amount of energy the cardiac muscle has to expend during systole to expel the same quantity of blood. In the first place, the relations borne by changes in circumference of a sphere to its cubic contents, show that a ventricle with a circumference of ten inches, if it contract up one inch during systole, will expel four times as much blood as a ventricle of five inches in circumference contracting up the same amount. Or, to put the matter conversely, the withdrawal of such a quantity of the contents of a sphere whose circumference is five inches, sufficient to reduce that circumference by an inch, will cause a reduction of only a quarter of an inch of the circumference when the circumference is originally ten inches. In other words, the larger the ventricle during diastole the less it will have to contract up during systole in order to expel a given quantity of blood, therefore the less work will the cardiac muscle have to do. In this connection, too, it must also be remembered that within certain definite limits the more cardiac muscle is stretched, the more is its irritability and its power of doing work increased.

But on considering the relation of changes of the circumference to the strain thrown upon the ventricular walls during contraction, in other words, the resistance to contraction, it is found that dilatation of the ventricle increases the work of the cardiac muscle to a marked extent. The strain on the walls of a hollow spherical muscle increases uniformly with the circumference, therefore the resistance to contraction of the ventricular walls becomes greater as the circumference is increased, and the more the ventricles dilate the more work will their muscular walls have to do to expel the same quantity of blood against the same pressure into the pulmonary artery or aorta.

It becomes clear, then, that for any given ventricle there is a particular diastolic circumference at which the ventricle will do



its work with a minimal expenditure of energy, and that if the circumference be increased by transfusion or lessened by venesection, the work of the heart will be uselessly increased. On the other hand, if the ventricle is too small to work economically, transfusion will help it, and if too dilated, venesection will have the same result. It is clear, too, that it is the nutritive condition of the cardiac muscle which will determine the exact circumference at which that particular ventricle will act to best advantage, and that, therefore, venesection or the reduction of the diastolic circumference of the ventricles can be employed usefully for the benefit of the heart only when it is certain that the ventricles are too dilated to be working economically. The clinical evidence of this condition is seen in a rapid, weak, often irregular, pulse of low tension, a labouring and dilated right ventricle and backward pressure along the systemic veins, a condition frequently observable in severe cases of pneumonia, asthma, bronchitis, and emphysema, of mitral or aortic disease, of uræmia, or of any other condition in which from any cause the cardiac muscle is rendered unable to perform the amount of work necessary to carry on the circulation. In any of these, venesection, by reducing the residual blood and consequently the diastolic side of the right ventricle, may lead to marked improvement of the circulation as a whole. In extreme cases of cardiac dilatation venesection may be an almost necessary preliminary to enable the overstretched muscle to respond to digitalis, strychnine, or other cardiac stimulants.

Venesection is also performed for the relief of the pain of thoracic aneurysm. How it brings about this result it is impossible to explain, since it is not obvious how venesection can alter either the volume of the aneurysm itself or the circulatory condition of the surrounding structures.

Turning now to transfusion, the cases in which it is most frequently performed are those which are often called indifferently cases of shock or collapse; but shock and collapse are two conditions which differ fundamentally in their pathology and in the treatment they require. I wish firstly, therefore, to deal with this point.

A person in a condition of shock, such as a man who has been kicked in the "wind" or the testicles, may superficially look very like a person who is in a condition of collapse following a severe hæmorrhage; in both the surface is cold and anæmic, the respirations shallow, quick and often irregular, the pulse of low tension, frequent and small. But there is this fundamental difference between the two, that the case of collapse has lost a large quantity of fluid from the vascular system and the case of shock has not.

What, then, is the pathology of *shock*? It may be defined as a reflex partial inhibition or paresis of the central nervous system, affecting especially the vasomotor but also the cardio-inhibitory and respiratory centres. The afferent impulses reaching these centres may be started either in the brain or at the periphery, and their action in inhibiting rather than exciting the action of the centres is an example of the rule that whilst weak stimuli may exalt, stronger stimuli may diminish the activity of any given part of the central nervous system.

The symptoms of shock are largely due to the fact that the vasomotor centre being temporarily inhibited, the small arteries, and to a less extent the veins, throughout the body and especially in the splanchnic area, lose their tone and dilate widely; a large proportion of the blood collects in the abdominal veins, and consequently the arterial blood-pressure falls, and the body generally is deprived of a proper supply of blood. In shock, then, there is no loss of fluid from the vascular system, therefore the specific gravity of the blood remains practically normal, but the patient has, so to speak, bled into his abdominal veins. The circulation through the two most important organs in the body, the brain and the heart, is carried on and regulated solely by the difference between the blood-pressure in the aorta and in the big veins near the heart. Owing to the great fall of arterial blood-pressure in shock, accompanied as it is by a relatively insignificant fall of venous pressure, the circulation through both of these organs suffers severely, and the failure of the cerebral circulation may even prove fatal.

For the treatment of shock, the obvious indication is to raise the arterial blood-pressure, and it has now to be considered whether

transfusion is a suitable means of doing so, or, if not, what other methods can be adopted. If salt solution be transfused under pressure into the vein of a patient in a condition of shock, it will for the time increase the intake of fluid by the heart, and consequently its output into the arteries; this will for a time raise the arterial blood-pressure, and consequently improve the coronary and cerebral circulations; the patient will feel better, and the pulse will be stronger. But the majority of the fluid injected will find its way through the dilated arteries into the abdominal veins and accumulate there, leaving the patient in a short time very much where he was before; as a matter of fact, however, the injection of a pint of fluid will cause a slight, but inadequate, permanent rise of arterial blood-pressure. If no more be injected, even the fluid introduced will not remain for more than perhaps an hour circulating within the blood-vessels, because it will pass out of the capillaries and be taken up by the tissues. The tissues generally, and especially the muscles, make every effort to keep the quantity of fluid circulating in the blood-vessels constant, and either take up fluid from the blood, or give up fluid to the blood, as the case may be.

In order to produce a permanent and adequate rise of arterial blood-pressure in shock, it would be necessary to transfuse very large quantities of fluid in a severe case, and to this there are several objections. It cannot be a good thing to water down the blood unless it is absolutely necessary; but a far more important objection to these large injections is, that owing to inhibition of the cardio-inhibitory centre in shock, the protective action of the vagus is removed from the heart, and it would be easy to produce engorgement and acute dilatation of the right side by injecting fluid in large quantities. Further, as has been already pointed out, it is by no means certain that it is possible by injections, no matter how large, to raise the arterial pressure to anything like the normal so long as the vasomotor paresis lasts.

How, then, is shock to be treated? In other words, how is this undue quantity of blood, stagnating in the abdominal and other veins, to be got out of the veins into the arteries; and how is it to be prevented from accumulating in the veins

again? A few experiments will make this clear. If a tame rabbit be held up by its ears feet downwards, it rapidly becomes unconscious and dies in about half-an-hour from failure of the cerebral and coronary circulations. The blood in the veins has, under the force of gravity, accumulated in the abdominal veins, just as it does in shock, and with the same results. This is prevented in the case of the rabbit by firmly bandaging the abdomen before holding it up by the ears. Again, in a dog, if the spinal cord be cut high up, so as to remove the action of the vasomotor centre and paralyse the ribs, leaving the diaphragm acting, as long as the animal is in the horizontal position his circulation is adequately maintained, but directly he is placed in the feet-down position it fails. This can be prevented either by compression of the abdomen or by artificial respiration. The body, then, has two principal mechanisms for preventing the blood from accumulating in the abdominal veins: (1) the vasomotor centre, which by its action constricts the small blood-vessels and so keeps up the arterial blood-pressure, and an excess of blood out of the abdominal veins, and (2) the respiratory centre, which by inspiration pumps and sucks the blood out of the abdomen into the lungs, and by expiration forces the blood out of the lungs into the arteries.

In shock, the vasomotor and respiratory centres are acting very feebly, and hence the symptoms; therefore, to remove the symptoms the actions of the centres must be replaced artificially in some such way as the following: (1) Perform artificial respiration in a severe case; its action has been explained above. (2) Place the patient with his head slightly lower than his feet; this will allow the force of gravity to help the return of blood along the veins from the abdomen. (3) Firmly bandage the abdomen, and if necessary the limbs. This will empty the veins into the heart and arteries, and by compressing the veins will prevent blood from re-accumulating there, and to this extent will replace the action of the vasomotor centre. (4) Stimulate the heart muscle by injections of strychnine and digitalis to perform an extra amount of work and pump the blood from the veins into the arteries. (5) Artificially warm the patient, because during shock

the production of heat by the body is decreased and the loss of heat from the body is increased, both of which lower the temperature of the body and consequently the recuperative power of the central nervous system. In short, the rational treatment of shock is, not to put more fluid into circulation, but to re-arrange the distribution of that which is already there.

The symptoms of *collapse* are due primarily to excessive loss from the vascular system of fluid, whether it be loss of blood as a whole or only of the watery part of the blood. It is not meant that in any given case bacterial and other poisons may not help in causing the symptoms, but simply that the fundamental cause of the symptoms is the one named.

The simplest cause of collapse is excessive hæmorrhage. Directly the hæmorrhage begins, the tissues begin to make good the loss of fluid from the circulation by passing water from themselves into the blood-vessels; consequently it is found that the blood begins to become more watery at once and its specific gravity to fall, whilst that of the tissues correspondingly rises. This effort of the tissues to keep constant the quantity of fluid in circulation by giving up water to the blood clearly points out that such cases should be treated by transfusion. In all cases of severe hæmorrhage the time to transfuse is directly the bleeding has been controlled. It is a well-known fact that cases which have lost considerable quantities of blood, but short of becoming at once profoundly collapsed, may appear for some hours to be going on satisfactorily, then they suddenly become collapsed and die. In them the supply of water from the tissues, together with increased action of the vasomotor centre, suffice for a time to maintain the circulation, but as soon as that source runs dry, and the vasomotor centre becomes exhausted by its efforts to keep up the blood-pressure, the symptoms of collapse set in and the circulation fails. Such accidents might probably be prevented by early transfusion, or by copious enemata of salt solution given as precautionary measures without waiting for symptoms of collapse.

In cases of slow continuous oozing of blood from the gums or other mucous membrane, it may be necessary to transfuse the

patient in order to keep him alive, although the hæmorrhage has not been stopped. In such a case it is simply a choice of evils, and the lesser, whichever it is, must be chosen.

In all cases of severe internal hæmorrhage, such as hæmoptysis, hæmatemesis, ruptured liver or spleen, or hæmorrhage from intestinal ulceration, as long as the bleeding vessel remains untied, immediate transfusion is certainly contra-indicated. When, however, there is reason to believe that the bleeding has ceased completely, and the patient is still dangerously collapsed, it might be wise to transfuse him cautiously, per rectum or subcutaneously, using small quantities at a time.

In many cases of collapse, however, the way in which fluid is lost from the vascular system is not so obvious at first sight as in the case of hæmorrhage. For instance, in cases of burn or scald it is a familiar fact that the prognosis is determined, not so much by the degree as by the area involved. Thus, a patient with one finger badly charred by fire, and another scalded slightly all over the body, are both at first in a condition of shock. The patient with the severely burned finger comes out from the condition of shock and recovers; the scalded patient may or may not recover temporarily from the shock, but passes gradually into a condition of collapse and die. Again, a patient has a blow in the abdomen which ruptures his gut, he may recover from the initial shock and even keep about for a day, feeling comparatively well, then he passes into a condition of collapse. It must now be asked, how has such a patient, and the one who was scalded, lost fluid from their circulation and become collapsed, and what has been happening to their circulation between the initial shock and the subsequent collapse?

Whenever a tissue is damaged, whether mechanically, chemically, or by inflammation, it becomes œdematous with fluid taken from the vascular system. In such patients as the ones considered, three distinct stages in the production of a fatal collapse can be distinguished: (1) In the first stage fluid is rapidly poured out into the damaged tissue from the blood-vessels, but the quantity of fluid in circulation, and therefore the specific gravity of the blood, remains normal because an equal quantity

of water is passing into the blood from the uninjured tissues. (2) During the second stage more fluid is passing into the injured tissues than can be got from the uninjured ones, therefore the specific gravity of the blood begins to rise; in other words, there is now less than a normal quantity of water in the total circulating blood. For a time this condition does not affect the blood-pressure and pulse because it is temporarily compensated for by vaso-constriction of blood-vessels. Clinically such a patient would not be collapsed in appearance, nor would his pulse indicate his danger. (3) In the third stage, the drain of fluid into the damaged tissues still goes on, the specific gravity of the blood rises continuously until it may be  $14^{\circ}$  or more above the normal, the vasomotor centre can no longer keep up the arterial blood-pressure, which falls progressively till the death of the patient from failure of the cerebral and coronary circulations. It has been found in fatal cases of collapse that as much as a third of the total water in the blood has left the blood-vessels and been taken up by the damaged tissues. This last stage of collapse, is of course, well marked on the pulse, and the shrunken appearance of the flesh shows how completely all possible fluid had been withdrawn from the tissues. It is very important to note that this final stage of circulatory failure may set in with great suddenness and the patient be dead before anything can be done for him.

Collapse may develop in exactly the same way from the continued loss of fluid by severe vomiting and diarrhœa, as seen in cholera, the summer diarrhœa of infants, ulcerative colitis, uræmia, in cases of irritant poisoning, and many other like conditions.

But enough has been said to make several points clear.

(1). Firstly, that in cases of possible extensive injury to tissues, such as blows in the abdomen, when it is often difficult to diagnose between shock and collapse, or after the shock has passed off to determine whether or not there is any injury to tissue, internal hæmorrhage or commencing peritonitis, an examination of the specific gravity of the blood may give reliable information hours before clinical symptoms appear. It is only necessary to remember that in uncomplicated shock the specific gravity of the

blood is not materially altered, but if it is, that it is slightly lowered; that hæmorrhage lowers the specific gravity proportionately to its extent, whilst injury to tissues without material hæmorrhage raises it.

(2). Secondly, that before severe operations, especially before long abdominal operations, or operations entailing much handling, tearing or damaging of tissues, it is a mistake to starve the patient of fluids. It may be advisable to cut off fluid by the mouth for fear of vomiting, but there cannot be the same objection to giving the patient plenty of fluid per rectum. It has been objected that absorption of large quantities of fluid, by producing a watery condition of the blood, would lead to increased hæmorrhage at the operation, but this objection has no foundation in fact. Within an hour of its absorption the excess of water will have been removed from the blood and largely stored in the tissues, where it will form an increased reserve of fluid to be drawn upon to make good loss of blood or water from the circulation.

(3). That all operations, especially abdominal operations, should be performed as rapidly and with as little handling and exposure of the tissues as possible. It has been found that at the end of a laparotomy lasting an hour the specific gravity of the blood may have risen as much as seven degrees, and that, other things being equal, the extent of the rise is directly proportional to the length of the operation. It also follows that the less tissue is damaged the better, and that the irrigation of tissues at the site of operation with irritating antiseptics is bad.

(4). That after any operation or extensive injury, the patient is put to bed in a condition consisting partly of shock and partly of collapse, the degree of collapse depending on either the loss of blood or the amount of tissue damage on the one hand, and on the available supply of water in the tissues on the other. Shock is not often a progressive condition, but collapse frequently is, and a rapidly fatal stage of collapse is liable to set in somewhat suddenly at the end of about twelve or twenty-four hours after a severe operation. It would seem wise, therefore, during the first few hours after such an operation or an injury, when the patient can take very little fluid by the mouth, to give plenty of fluid by



the rectum where possible ; it is, too, during these early hours that the specific gravity of the blood should be carefully watched as an indication for early transfusion.

(5). That all cases of collapse are pre-eminently suitable for treatment by transfusion. In cases of hæmorrhage this should be done directly the bleeding has been controlled, in other cases directly the specific gravity of the blood begins to rise. Collapse, unlike shock, comes on slowly and insidiously at first, and if treatment is delayed until the patient is collapsed clinically, it has been put off until he is within measurable distance of death.

In collapse, owing to the fall in arterial blood-pressure, the secretion of urine is greatly decreased, and may even be arrested altogether. In septic peritonitis, or intestinal obstruction, and many other conditions in which there is, in addition to the cause of the collapse, absorption of toxic material as well, this decreased power of eliminating poisons by the kidneys is very serious. If such a patient be transfused the poison in his blood is temporarily diluted, but above all, by raising his arterial blood-pressure, his kidneys are enabled to pass out larger quantities of poison.

Several questions now arise :—

(a) How can you rapidly and repeatedly take the specific gravity of a patient's blood? The simplest method is that of Hammerschlag: chloroform is heavier than blood; benzol, or xylol, is lighter; mix in a urinometer glass such quantities of the two that the specific gravity of the mixture taken by a urinometer is 1,060 which is equal to that of normal blood. Puncture the ear, draw a drop of blood into the tube of a Thoma-Zeiss pipette, or any other capillary tube, and blow it out again into the chloroform, benzol or xylol mixture. The blood does not mix at all with these liquids, but floats in them like a red bead. If it sinks to the bottom, add chloroform; if it rises to the top, add benzol, or xylol, until finally the drop of blood remains stationary in the body of the liquid, showing that its specific gravity is just that of the surrounding mixture. Then take the specific gravity of the liquid with the urinometer float, and that is the specific gravity of the drop of blood that floated in it. Filter the fluids, and they are ready for use again.

(b) A second not unimportant point to discuss is whether sodium chloride is the best salt to use, and whether one drachm of it in a pint of tap-water is the right proportion. The main point in favour of sodium chloride is that it is always obtainable, and that in weak solutions it is practically harmless. By using tap-water some calcium salt is also injected, and this is of importance in maintaining the tone of the heart muscle. On theoretical grounds it might be advisable to add a small dose of potassium salt, but practically it would be wiser to omit it, as in excessive doses all potassium salts are highly poisonous.

On the other hand, it is equally true that the proportion of one drachm of sodium chloride to a pint of water is not the right one for injection into human beings, and this is of more than theoretical importance. Normally the blood inside the capillary, and the lymph outside it, have the same percentage of the same salts in solution and are therefore said to be isotonic, *i.e.*, they have the same strength of attraction for water, and consequently neither the blood nor the lymph will attract or give up water to the other. It is clear, then, that a patient ought to be transfused with a fluid which is isotonic with his blood and lymph. Sodium chloride one drachm to a pint is isotonic with frog's blood, but is too weak to be isotonic with human blood; the proportion ought to be one and a half drachms to a pint.

The principal effects of transfusing with too weak a salt solution are to enable the tissues generally to attract more water out of the blood-vessels, which is the very thing the transfusion is meant to correct, and also to cause the breaking up of a certain number of the red blood corpuscles, which if preventible, is undesirable.

(c) What is the indication of how much fluid is to be at once injected and whether it is to be repeated or not? In all cases of collapse due to hæmorrhage in which the bleeding has been controlled, the indication is to immediately transfuse at least as much fluid as the patient has lost blood. If besides the mere hæmorrhage there are damaged tissues as well, which will take up fluid from the vascular system, an extra quantity must be

injected. It may safely be said that it is easier to inject too little than too much.

If the collapse is due to loss of fluid, as opposed to loss of blood, then in the specific gravity of the blood there is a reliable indication of how much and how often to transfuse. Take for instance a bad case of scald. Examination will show that the specific gravity of the blood is rising or is already high above the normal, transfusion will bring down the specific gravity of the blood again to normal, in other words, increase the quantity of fluid in the circulation. The patient's general condition may appear considerably improved, but only for a time, again the profound collapse sets in. On again examining the specific gravity of the blood, even at the end of an hour from the first injection of fluid, it will be found that fluid is still leaving the vascular system rapidly because the specific gravity of the blood is rising again and may even be higher than before the first injection; therefore transfuse again. In experiments on animals it has been found that this process may have to be repeated half-a-dozen times, but that after each injection the specific gravity of the blood rises more slowly, until finally it ceases to rise after the transfusion, but remains about normal, then enough fluid has been injected. The explanation is clear. The blood to start with is inspissated, the uninjured tissues dry, and the injured tissues with their capillary walls more than normally permeable and ready to take up all the fluid they can hold. Of the first injection most of the fluid rapidly passes into the injured tissues, leaving the condition but little improved. The injections must be continued until the injured tissues have taken up all the fluid they can hold, until the uninjured tissues have replenished their store of water, and then whatever quantity of fluid is left in the circulation will remain there, and when the specific gravity of the blood has been finally brought back to normal, the proper quantity of fluid will certainly be left in the circulation.

Since in cases of severe collapse one transfusion may be perfectly useless, it may well be asked if there is any danger in

such repeated transfusions. The answer is, certainly not, provided that the injections are made slowly and at a low pressure. The blood in such cases is inspissated through loss of water with salts in solution; a certain quantity of proteid will also have left the blood and have gone into the damaged tissues as lymph; but that would go anyhow, whether the patient were transfused or not. The bulk of the proteid with all the red blood corpuscles and hæmoglobin will still be in the circulation, so that at the end of the series of transfusions, the blood will be practically normal in quantity and only differing in quality in having a little less proteid and salts than it should.

Besides cases of shock or collapse, or a combination of the two, there are many other conditions in which transfusion has either been tried or might appear to be a possible method of raising the arterial blood-pressure, or of combating toxic conditions of the blood.

To take first, cases of cardiac failure, whether due primarily to disease of the muscle, arteries, nerves, or valves of the heart, or to causes peripheral to the heart itself, such as continuously high arterial blood-pressure, general blood-diseases, cardiac poisons, etc. In nearly all such cases the arterial blood-pressure is low, and the question arises: Is transfusion a suitable method of raising it?

*Cardiac failure* is synonymous with inability of the heart muscle to perform the amount of work it is called upon to do. It is also true that cardiac failure does not lead to any very serious disturbance of the systemic circulation so long as the tricuspid valve remains competent. In other words, before the systemic circulation can be seriously disturbed by cardiac failure, the muscle of the right ventricle must fail. Whenever the right ventricle, like the left, is called upon to do an extra quantity of work, the first effect of this extra work is to increase the quantity of residual blood, *i.e.*, the quantity of blood left in the ventricle at the end of its systole; consequently, in order to accommodate the blood coming in during diastole, the cavity has to dilate. This dilatation must lead to a stretching and thinning out of the ventricular muscle, and therefore to an increased resistance to

contraction of its muscular wall ; in other words, in order now to send out the same quantity of blood the ventricular muscle will have to exert more force and expend more energy. As long as the muscle of the right ventricle can contract with this necessary increase of force, the circulation is adequately maintained ; but directly the muscle begins to fail, a further dilatation of the cavity takes place, until ultimately the tricuspid valve becomes incompetent. Then the general circulatory disturbance of cardiac failure begins, the right ventricle being unable to pump out as much blood as it receives from the large veins, the venous pressure rises, the right ventricle is dilated still further, and the whole venous system engorged with blood. On the other hand, less blood is now being pumped by the left ventricle into the aorta, the arteries are relatively empty, and the arterial blood-pressure falls.

However desirable it may be to get more blood into the arteries and so improve the coronary and cerebral circulations in particular, it is manifestly absurd to attempt this by offering the right ventricle more fluid when it is already labouring with more than it can deal with. It may, therefore, be laid down as a general rule that in all cases of circulatory disturbance due primarily to cardiac failure transfusion is contra-indicated.

Venesection, on the other hand, is strongly indicated in all cases of right-sided engorgement. The removal of twelve or more ounces of blood from a vein, causes a corresponding decrease of the quantity of blood offered to the right ventricle, hence of its residual blood, of its condition of dilatation, and consequently of the amount of energy it will require to expend in pumping the proper quantity of blood into the left ventricle. In this way venesection, by helping the right ventricle, may raise the arterial blood-pressure and improve the circulation as a whole.

In passing, it may be pointed out that the position of orthopnea when assumed by patients with an engorged right side is really, as it were, an attempt to perform venesection on themselves. By their more or less perpendicular position they allow the force of gravity to act on the column of blood in their inferior vena cava, and so keep back in their abdominal veins blood which would

otherwise only help to dilate still further their right ventricle. Again, their respiratory centre tries to make good the weakness of their right ventricle by a forcible action of the respiratory pump, which, during inspiration, will drive some blood through the distended right ventricle into the lungs, and will by expiration empty the lungs into the left heart.

*Uræmia* is the name given to a group of symptoms which arise in the course of many renal diseases, whether due to inflammation, destruction by various morbid processes, or damage by poisons, etc. Whether in all cases the cause of the symptoms is exactly the same or not is uncertain, but presumably in most cases the symptoms are due to the retention in the body of one or more substances which have accumulated in the blood and tissues to a poisonous extent owing to their not having been excreted sufficiently rapidly by the kidneys. Transfusion has frequently been tried in the treatment of uræmia with the idea of diluting the poison in circulation, and so of allaying the symptoms for a time at any rate. The results of transfusion have been very variable; in some cases the symptoms have rapidly passed off, in others no apparent effect has been produced, and in others death has occurred very soon after the transfusion. It is worth discussing, therefore, whether it is possible to foretell with any certainty or not in which cases transfusion is likely to do good and in which not.

For this purpose the cases of uræmia must be divided into two groups. I. Those with an abnormally low arterial blood-pressure. II. Those in which the arterial blood-pressure is equal to or above the normal.

I. Cases with an abnormally low arterial blood-pressure. There are two main causes of a considerable fall in arterial blood-pressure in renal disease. (*a*) Cardiac failure and (*b*) collapse, and they will be dealt with in that order.

(*a*.) Several chronic forms of kidney disease are associated, in their early stages at least, with a high arterial blood-pressure; this must throw extra work on the heart, and as long as the heart can meet it and keep up the arterial pressure, the kidneys excrete moderately well and the patient does not become uræmic. Sooner or later, however, the cardiac muscle may begin to fail, the

dilated and hypertrophied heart dilates still further until the tricuspid valve becomes incompetent, and the circulation as a whole fails. The secretion of urine depends very largely upon the rate of flow of blood through the kidneys, which is itself determined by and varies directly as the fall in blood-pressure between the renal artery and the vein. In cardiac failure the difference in the blood-pressure between these two points, and consequently the secretion of urine is greatly diminished; it is in this condition that the majority of such patients become uræmic. In such a case, either before or after uræmia has set in, it is clear that one of the primary objects for treatment is the cardiac failure, and that for this, as has been explained before, venesection and digitalis are indicated and not transfusion; in fact, transfusion would be dangerous.

(b.) The second cause of low arterial blood-pressure is collapse. The chronic or subacute variety of uræmia is not infrequently of a gastro-intestinal type; it begins with intense and persistent vomiting and diarrhœa. These may go on for days or weeks; then follow muscular twitchings, cramps, dyspnœa, and delirium, passing into drowsiness and coma. The vomiting may be of central origin, but it seems likely that both it and the diarrhœa are in part due to the excretion of toxic material with water by the mucous membrane of the alimentary canal compensatory to the kidneys; and it is these cases especially which show post-mortem inflammation or ulceration of the large intestine. Such patients lose more fluid from their vascular system than, owing to vomiting, they can make good, and they gradually pass into a condition which is fundamentally one of collapse. Their arterial blood-pressure is low; the secretion of urine is almost suppressed, all possible water having been abstracted from their tissues. They are not œdematous, thus forming an exception to the general rule—the less urine the more œdema; their blood becomes inspissated and consequently the toxin in it must thereby be concentrated, in fact it is almost fair to say that the collapse determines the uræmia. To transfuse such a patient, frequently if necessary, is to dilute the poison in his blood, to raise his arterial blood-pressure and to enable what is left of his kidneys to start secreting again,

and if done in time it might even ward off an attack of uræmia. Exactly the same is true of patients suffering from cholera infantum, cholera nostras, etc.

II. Cases with arterial blood-pressure—not less than normal, or abnormally high. It will be necessary to divide this class into two groups of cases, and to discuss them separately, (1) those in which the high arterial tension is associated with no marked cardio-vascular changes, and (2) those in which cardio-vascular changes are present.

Group (1) is practically confined to cases of acute nephritis in previously healthy kidneys and of puerperal eclampsia. In acute nephritis the high arterial tension begins at the very onset of the disease, and is presumably due to the action of some poison in the blood on the vasomotor centre, and perhaps also directly on the peripheral arteries; it is not due to structural changes in the arteries, which are practically sound, as is also the heart. The same is true of eclampsia, the blood-pressure which may have been higher than normal throughout pregnancy, becomes very high before the fits set in. Before discussing the suitability of transfusion in the treatment of acute nephritis, or eclampsia, it is necessary to try and determine the state of the renal circulation in these conditions and its relation to the urinary changes.

In the kidney there are two excretory mechanisms to some extent independent of each other, namely, the glomerulus for filtering off water and salts in solution from the blood, and the tubular epithelium which picks up from the blood and passes on the organic constituents of the urine; an upset of the former mechanism will chiefly affect the quantity of the urine excreted, and a derangement of the latter its organic contents. It is necessary to bear this in mind in attempting an explanation of the observed urinary changes.

In animals, electrical stimulation of the spinal cord, and consequent vaso-constriction of almost every arteriole in the body, raises the arterial blood-pressure enormously, but in spite of this the secretion of urine ceases owing to the vaso-constriction of the renal blood-vessels; post-mortem examination shows anæmic



kidneys. This condition of pale bloodless kidneys without other obvious change has actually been found post-mortem in some cases of fatal eclampsia. Granted, therefore, a toxic condition of the blood in pregnancy which is capable of producing vaso-constriction through the vasomotor centre, a patient might in a few cases be rapidly killed by an accumulation of the poison due to anuria. Should such a condition exist, and were it possible to diagnose it clinically by the absence of blood or albumen in the urine, it is clear that transfusion, by diluting the circulating poison, and consequently the renal vaso-constriction, might offer a reasonable line of treatment.

In most cases of fatal eclampsia and of uræmia in acute nephritis, the kidney is not anæmic but engorged with blood, with acute inflammation and œdema of the interstitial tissue and degeneration and desquamation of the tubal epithelium in varying proportions.

So many diseases are known in which primary acute nephritis, not an acute attack of inflammation in an already chronically diseased organ, is clearly due to excretion by the kidneys of large quantities of various toxins, that it may well be doubted whether acute nephritis is ever a disease originating primarily in the kidneys. Granted that the excretions of toxins can set up inflammation of the kidneys, there still remains for explanation the blood-dripping organ and the greatly diminished secretion of water as well as of solids. If the great vascularity of the kidney were due to dilatation of its arterioles, then with the high blood-pressure there ought to be a plentiful filtration of water through the glomeruli, but this is not so. On the other hand, if the diminished filtration of water from the glomeruli, in spite of the high blood-pressure, is due to constriction of the renal arterioles, then it is impossible to account for the vascular engorgement of the kidney. It is more than likely that the renal arterioles do dilate when the kidney becomes inflamed, but as in other inflamed organs, it is not arterial dilatation but strangulation of the veins which causes the engorgement with blood. Strangulation of the veins in any inflamed organ is brought about in the following way—the arteries,

capillaries, and veins are bathed in transuded fluid, the fluid which leaves the inflamed capillaries will, if a free outflow for it be absent, after a time attain a pressure near that ruling in the capillaries, and higher than the venous pressure. The veins will therefore collapse, venous obstruction will be produced, the capillary pressure and transudation will be higher than ever, so that there is produced a vicious circle of events tending continually to increase the œdema, the venous obstruction, and the vascular engorgement of the part. In fact, the flow of blood through the inflamed kidneys may almost cease, hence the diminution in the quantity of urine secreted.

There is another fact which makes it likely that the cause of the failure of the secretion of urine in these cases is a vascular one, and that is the rapidity with which, when once the kidney has begun to right itself, its powers of secreting urine return. Allowing, then, that the above represents the condition of the circulation of the kidneys in uræmia during acute nephritis, and that the restoration of a rapid circulation through the kidney by the removal of venous strangulation is the change to be desired, is transfusion and the consequent raising of the general venous blood-pressure likely to improve matters? The answer would seem to be—certainly not.

(2.) In chronic nephritis, in which cardio-vascular changes are present and the arterial blood-pressure is high, it is necessary to consider, (a) do the cardio-vascular changes contra-indicate transfusion for uræmic symptoms? Since the arterial blood-pressure is high, the heart must be acting fairly well to keep up the blood-pressure, therefore transfusion would not be contra-indicated on its account. With regard to the arterial change, transfusion, by producing for a few hours a condition of hydræmic plethora, would certainly for that time largely increase the blood-pressure in the small vessels of the brain, as elsewhere, and might therefore possibly be the existing cause of a cerebral hæmorrhage.

(b) Is transfusion likely to improve the secretory powers of the kidneys? In cases of uræmia occurring in chronic nephritis in which the arterial blood-pressure is normal or above it, the secretion of urine is in one of two conditions (i.) it may be greatly

reduced in quantity, or (ii.) it may be normal or even above normal in quantity but poor in urinary solids.

The reduction of urine in the one class of case cannot be due to cardiac failure or to collapse, both of which are accompanied by an abnormally low arterial blood-pressure, but to the inflammation of the kidney substance interfering with the local circulation, a condition which has already been explained, transfusion is not likely to benefit. Cases of uræmia in which the secretion of urine is not reduced are chiefly cases with granular kidneys. The fact that the secretion of water is not reduced must mean that the vascular supply of the kidneys is good, but that the amount of renal epithelium left is insufficient to keep the blood in a non-poisonous state. Since transfusion is not wanted to improve the circulation, but could only be of use in temporarily reducing the toxic condition of the blood, is it likely that it could produce a successful result so far as it concerns any given attack of uræmia in a case of granular kidneys? The destruction of kidney substance in renal cirrhosis is generally so slow that there must be a considerable period of time during which a patient may exist with only just enough kidney substance left to keep him going; any material increase in the work it is required to do may be more than the kidney can manage. It is during this period of apparent health that uræmia often sets in with considerable suddenness in cases of granular kidney, and in a large proportion of such cases there is found, post-mortem, in some part of the body an early septic inflammation, the toxic material from which constitutes the extra secretory work which the kidneys are unable to perform, and determines the attack of uræmia. It is notorious how serious any inflammation is in such a case, but it seems probable that transfusion, repeated at intervals, would be one of the most effective methods of possibly tiding the patient over the time during which his kidneys were unable to excrete toxic material as fast as it was being turned into the blood.

There are two other conditions in many ways similar to uræmia, namely, cholæmia and diabetic coma, in which the symptoms are due to the accumulation in the blood of poisons produced in the body more rapidly than they can be passed out

by the kidneys. In cholæmia the poison is very probably one of the compounds of ammonia, the carbonate, carbamate or lactate, all of which are extremely poisonous, and one or more of which certainly leaves the muscles to be converted by the liver into the innocuous body urea. When this function of the liver begins to fail, symptoms of poisoning soon set in.

In diabetic coma the exact nature and seat of formation of the poison are less known. Both conditions are nearly always fatal, but transfusion, by diluting the poison and encouraging its excretion through the kidneys, has in many cases led to a marked but temporary improvement and return to consciousness.

The foregoing considerations have shown that transfusion can produce certain profound changes in the circulation, it must therefore have definite indications and contra-indications; some of these are clear, but many others will only be brought to light by careful selection of the cases in which it is employed and by observation of the exact effects it produces upon each of them. It may well be doubted whether transfusion, intravenous, subcutaneous and rectal, are used to anything like the extent they deserve, and this has probably arisen from it not being generally appreciated how enormously important it is to keep the quantity of fluid in the circulation up to the normal, and how many patients really die from sheer lack of it. On the other hand, transfusion is no panacea for circulatory failures, many are certainly aggravated by it, and, like venesection, it might easily be unjustly condemned and go out of fashion simply because it was used ignorantly and indiscriminately.

# NOTES ON TUBERCULAR DISEASE OF THE LYMPHATIC GLANDS IN THE NECK.

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By C. J. HARNETT, M.D.

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My apology for writing this paper must be, that having myself suffered from the scanty treatment that the subject receives in the ordinary text-books on Surgery, I am encouraged to hope that my notes may prove of some slight value to those, who, in the course of their practice, are called upon to treat this somewhat common disease.

I have based my remarks not only upon the results of private practice, but also upon the experience of eighteen months spent at the Royal Sea Bathing Hospital at Margate, during which period I was acting as Resident Surgeon to that Institution.

The great majority of the cases treated at this Infirmary are of a tubercular nature, and some two hundred cases of tubercular disease of the cervical lymphatic glands were treated in the wards during my term of residence.

As a preliminary, I propose giving the following classification of various types of the disease. Although fully aware of the danger of too hasty classification I have ventured to append the following scheme, because I have found it of considerable practical value in making notes of the various cases which have come under my care, and in enabling me to decide the main lines of treatment to be followed in individual instances.

It must, however, be borne in mind that I give this classification merely in a tentative manner, and that it is rarely that any two cases exactly resemble one another. At the same time there are certain main features which arrange themselves in different categories, and it is on these that my method is founded.

The types of the disease I have arranged under five headings, as follows :—

A. In this, the first class, the disease commences more or less acutely, usually in a subject with a history of tubercular trouble in the family. Commonly the glands on both sides of the neck are involved, those in the anterior triangles being most prominent. They rapidly attain a considerable size, are painful, are frequently the site of periadenitis, causing them to become very adherent to one another and to the surrounding structures; they break down early, and the patient's general health suffers in a marked degree.

In B. The disease comes on insidiously, usually without any apparent local cause. The child is out of health and "lumps are felt in the neck." The glands in the submaxillary and posterior triangles are perhaps those most frequently affected. They do not attain a great size, are discrete, and for the most part fairly movable. The patient suffers little inconvenience locally, the glands being painless or only slightly tender. Any intercurrent illness may, however, light up the disease, the glands enlarging, and eventually caseating with the formation of tuberculous abscesses.

C. In this the third type the enlargement is not infrequently entirely unilateral, and is usually far more marked on one side than the other. The disease attacks, in the first instance, the glands in the anterior triangle, those in other situations, however, subsequently becoming involved. The affected glands attain gradually to a considerable size. They are not so firmly adherent as those in Class A; and are, as a rule, painless except when the patient's health suffers from some other cause. Under such circumstances they are liable to become inflamed, the subsidence of the inflammation leaving the glands larger and more adherent.

D. This, the fourth type, is the form the disease often assumes when it shews itself in weakly, ill-nourished and neglected children. The disease is often accompanied by hypertrophy of the tonsils and the presence of adenoid vegetations. The glands are generally superficial, and may appear in any situation in the neck, perhaps most commonly in the posterior triangles. They do not become very large, but very early and when quite small break down and form little cysts containing thin liquid matter, which soon gives rise to subcutaneous abscesses, rapidly involving the skin.

E. To the foregoing I have added a fifth and last class, which is scarcely a type of the disease, but rather the result of neglect of any of the above. The glands are broken down, and abscesses, often deeply situated beneath the deep fascia, exist. These may have reached or even perforated the skin, so forming intractable sinuses.

Having thus given my provisional classification of the different types of the disease, I propose now to deal with the different methods of treatment, but before doing so a word or two as to the commonest known antecedents of the affection will not be out of place, as a recognition of these will provide the best means of successful preventive treatment, especially in the case of those who have any predisposition to tuberculosis.

Thus, perhaps, the most ordinary cases are those in which the disease follows simple inflammatory swelling, which in its turn is due to similar affections of the parts supplied by the terminal lymphatics. Examples of these are to be found in practically all forms of sore throat, particularly in the repeated attacks of tonsillitis to which the subjects of enlarged tonsils and adenoids are liable; in dental caries, stomatitis, otorrhœa, impetigo capitis, &c.

The glands become inflamed and swollen, they neither suppurate acutely nor entirely subside, but after some time has elapsed increase in size, other glands becoming enlarged. It is seldom possible to make a diagnosis of tubercular disease in the earlier stages. The persistence of the enlargement after the subsidence of the condition which caused its origin, engenders the suspicion

that the disease may be tubercular, and the suspicion approaches a certainty if the swelling persists for months, and glands enlarge in other situations.

These considerations emphasise the importance of careful treatment of such affections, affording a warning against falling into the error of attaching little importance to these ailments, trivial in themselves, yet of no inconsiderable moment in the case of children whose life-history or surroundings, or both, predispose them to tubercular mischief.

No matter to which type of the disease a particular case may belong, *the* most important factor in the treatment is the prompt insistence upon thoroughly hygienic surroundings. To ensure these the first step necessary is to remove the patient to the seaside.

In the choice of a health resort the following points should be borne in mind :—

Firstly. The air should be bracing and dry.

Secondly. The subsoil should be porous, and the vegetation scanty.

Lastly. It should be practicable for the patient to spend a considerable portion of each day out of doors.

The invigorating air of the East Coast is peculiarly suitable, and no place fulfils the above conditions more completely than Margate.

There the air is peculiarly bracing. The absorbent nature of the subsoil, the upper chalk, accounts for the fact that even during the wettest weather that raw, damp, penetrating cold which is so harmful to invalids is never experienced.

The country around is very sparsely wooded, and owing to its geographical position every wind, except that from the south-west, is a sea wind.

The mean winter temperature of Margate, contrary to popular impressions, is decidedly above that of London, and only two degrees lower than that of the warmest seaside resort in the United Kingdom, namely, Ventnor.

In the words of the late Professor Airey, Astronomer Royal, “ Margate has a larger number of hours of sunshine, a less



rainfall, and a more even temperature than any other seaside town in the United Kingdom." These features in the climate of this town help to account for the fact which experience has shown, viz., that in subjects of surgical tuberculosis a residence in Margate gives results which can be obtained nowhere else.

This was recognised as long ago as 1791, when after careful investigation by a committee of experts, Margate was chosen as the most suitable locality in which to found a hospital for the treatment of scrofula. Subsequent results obtained at this institution, the Royal Sea Bathing Hospital, have fully justified the choice.

It must be remembered that treatment by change of air, to be effectual, should be continued for long periods and frequently repeated.

The right hygienic surroundings having been secured, it will be found that in many cases supplementary treatment by the use of drugs will be unnecessary, and we have yet to discover a drug which exerts any specific action on this disease. Drugs, however, have their value mainly as a subsidiary means of improving the appetite.

Most of these patients are anæmic, and arsenic is the most suitable drug to use in its treatment, although its benefit is indirect and probably not due to any specific action. Many patients are thin, and most are improved by the administration of cod liver oil, which, on the whole, is the most valuable medicinal measure which we possess.

Of far more importance than medicinal treatment is the provision of an abundance of plain wholesome food, containing a plentiful supply of fat.

Attention should be paid to the house, particularly the sleeping apartment. This should have preferably a southern aspect, be lofty and well ventilated. The window should be constantly open, care being taken that the bed be not placed directly between the latter and the door or fireplace.

The choice of a living room is of minor importance, as it is essential that the patient should spend the greater part of each day out of doors. Even in wet weather this should be advised,

suitable clothing and the protection afforded by the ordinary seaside shelter being all that is required to render this beneficial. Woollen material should be worn next to the skin, and the healthy action of the latter, ensured by daily salt baths in the open sea during the summer months, or in suitable baths during the winter.

Every case, however, should be carefully watched. Should any tenderness or signs of inflammatory trouble arise, any but the mildest physical exercise should be prohibited, glycerine and belladonna applied, and the neck supported by firm bandaging over a collar of gamgee, or wood-wool, tissue. These measures usually result in a subsidence of the symptoms. Should, however, this not prove effectual it will be well to forbid all physical exercise, the patient being taken into the open air in a bath chair. In some cases, in spite of these precautions, the condition becomes more acute, the glands threatening to break down. In such instances absolute rest must be enjoined, and hot fomentations frequently applied, the patient being kept in bed. When once the glands have broken down nothing short of operative interference will be effectual. If inflammatory attacks of this nature become frequent the continuous use of supports to the neck, in the shape of firm bandaging over a collar of wood-wool tissue, or even the application of a gutta percha splint moulded to the neck and shoulders is advisable.

It would seem scarcely necessary to emphasize the fact that it is the greatest mistake to rub in applications to inflamed tuberculous glands, were it not that this appears to be a not uncommon practice. Rest is the principle to be aimed at, combined, where possible, with a continuance of the open air treatment.

Besides these hygienic and medicinal measures we have to consider the advisability of resort to operative interference. In discussing this question I shall assume that the patient is in a position to obtain the necessary hygienic surroundings; for, if not, as is too often the case with the poorer classes, operation can generally be recommended earlier and with more confidence.

There are certain cases in which one can say at once that operation is certainly not advisable—viz., where the glands have

only existed a short time, *i.e.*, a few months; are not breaking down, have not grown rapidly, and there is no marked deterioration of the general health.

There are other cases in which operative measures are imperative, *viz.*, where the glands have broken down, abscesses formed, or sinuses exist which show no tendency to heal.

There remains a large number in which the decision of the question is not so obvious.

While admitting that exceptions may be found to the following rules, I think they are of decided value in forming a conclusion.

If, then, the glands have not become very large, are not breaking down, and have existed for *less than two years*, hygienic measures should first be given a fair trial, and provided that these are efficiently carried out and continued for long periods, there is no doubt that in a large proportion, resolution will occur.

On the other hand, if the disease has existed for *over two years*, and the glands have attained a size to produce an obvious tumour, the patient may confidently be advised to have them removed, because it is practically certain that they will not subside under hygienic treatment alone, and extremely probable that they will eventually lead to the formation of abscesses and sinuses. Glands which have been enlarged for this time, however firm and free they may feel, have practically always undergone caseation.

From the foregoing remarks it will be gathered that cases which I have included in class A will seldom be cured without resort to operation. Hygienic measures may be tried, but unless they soon cause a cessation of the activity of the disease, they should not be prolonged alone, but excision should be early adopted. If when the case is first seen caseation has already occurred, it is important that operative measures should be undertaken without delay, otherwise considerable destruction of the skin may result, rendering it difficult to avoid the formation of disfiguring cicatrices.

The group B, on the contrary, if taken in hand early, will, in the majority of instances, be effectually cured by hygienic treatment alone, and it will only be a small minority which in spite of

such measures will go on to caseation, when, of course, operative interference will be necessary.

The treatment to be adopted in the cases classified under heading C will depend entirely on the stage at which the case is first seen. Those in which the disease has existed for over two years without obvious softening, form *the* most satisfactory cases for excision, for although the swelling will not disappear spontaneously, the disease is in a quiescent condition and excision will generally result in a permanent cure.

It is of the utmost importance that the subjects of the fourth type of the disease, D, should be at once removed into suitable climate and surroundings; and although surgical interference will be necessary, it is wise to defer such measures until such time as the child's health has commenced to improve.

Lastly, neglected cases included in class E will almost invariably require operative treatment. In some very long standing cases, however, there may be very little of the original disease left, and the septic sinuses will heal under antiseptic dressings alone, provided the patient be removed to suitable surroundings. In the treatment of discharging sinuses great care should be devoted to surgical cleanliness. Dressings should be abundant and frequently changed, the discharge never being allowed to soak through. These tedious cases will thoroughly repay the trouble thus taken.

Before offering general suggestions as to the nature and scope of the operation, which must necessarily depend largely upon the character of the case, I would draw attention to the following considerations :—

Firstly, tuberculous gland disease is very seldom in itself fatal; it is thus quite distinct from cancer, there being a tendency towards spontaneous cure.

On the other hand, it must be remembered that the disease may last a very long time, that suppuration may occur, and sinuses form, the patient being debarred from an active life for considerable periods, and although eventually a cure will result, very disfiguring scars may remain to tell the tale.

It is well also to recollect that, though of very infrequent occurrence, cases of tubercular pleurisy following this disease are not unknown.

Again, any local tuberculous centre is a possible source of general infection, although this event is a decidedly rare result of tubercular disease of the cervical glands.

Lastly, the scar from an operation wound may be, and by no means infrequently is, the seat of keloid formation, which leaves most unsightly marks.

It is therefore with the following objects that an operation is undertaken :—

1. To shorten the period of incapacitation of the patient.
2. To prevent deformity in the shape of unsightly cicatrices.
3. To remove the disfigurement caused by the presence of swollen glands.
4. To save the patient from the risk of—
  - a. Spread of the disease to other glands.
  - b. To other parts—pleura and lungs.
  - c. General tuberculous infection.

Although not great, it cannot be denied that every surgical operation is attended with a certain risk, and that for the removal of tuberculous glands from the neck forms no exception. Thus there is, of course, the usual small risk from the use of an anæsthetic. Owing to the intimate nature in which tuberculous glands frequently adhere to the large veins of the neck, the risk of severe hæmorrhage is perhaps greater than in most surgical operations of similar magnitude.

Attention to aseptic principles practically does away with that of septic infection. The possibility of entry of air into a large vein accidentally wounded must not be ignored.

Having decided that operative interference is necessary I will first consider those cases in which there are no existing sinuses or suppuration. In these excision is doubtless the correct operation. Here, however, I would emphasise the advantages to be derived from a preliminary period of entire rest. After two or three weeks in bed, under such hygienic conditions as I have described above, it will frequently be found that the patient's

general health has materially improved, the enlargement has greatly subsided and the glands have become less adherent. Especially does this apply to cases where the glands are much enlarged and firmly adherent.

As to the scope of the operation I think that the extensive operation advocated by Mr. Watson Cheyne in the *Lancet* for December 16th, 1899, is unnecessarily severe.

This operation, a complete extirpation of glands and fat in the area operated upon, necessitating in many instances removal of the internal jugular vein, in fact differing in no way from that adopted for malignant glands, naturally requires a very free exposure of the parts. To obtain the necessary degree of exposure, a free incision, and if there are glands in the posterior triangle, a vertical incision is required, in other words, an unsightly cicatrix is very likely indeed to result. There is no doubt whatever that the scar left from a vertical incision is always more unsightly, and far more frequently the seat of keloid formation than that left from an incision in the lines of natural cleavage of the skin. These latter run more or less in a transverse direction.

Seeing that one of the objects for which the operation is undertaken is to prevent or remove deformity, this alone is a very decided objection to such extensive operations. More especially does this apply when the patient is a young girl.

Again, provided a good result can be obtained, it is very important not to expose the patient to the shock necessarily associated with such severe proceedings.

In many cases diseased glands can be removed completely through quite small incisions made in a transverse direction. Large masses in the anterior triangle can be reached through a curved incision along a natural crease following the angle of the jaw. For the removal of large masses in the posterior triangle two or three transverse incisions can be used, and although the access to the glands is not so complete as when a long vertical incision is adopted, in most cases they will suffice. Only in the very worst cases, such for instance as those in which are found large masses of adherent glands extending beneath the sterno-mastoid, will it be necessary to use the long vertical incision. But

even here division of the latter muscle should be avoided, as it will greatly increase the risk of production of an unsightly cicatrix.

Without going into details, I may briefly indicate the nature of the operation. Having chosen the incision, this is carried down through the superficial structures until the nearest gland is exposed. The capsule of the latter is firmly seized with toothed forceps and firm traction put upon the gland; this is then gradually freed from its attachments by cutting lightly on to it (the gland), keeping away from its deep connections; in this way it, being partially freed, drawn out of the wound, and still cutting on to it, all but its deepest connections are severed, the final separation being accomplished by a blunt instrument or the fingers. (The important point to be observed in this part of the operation is, that the cutting must be on to the gland itself and well away from its deep attachments. Neglect of this simple precaution greatly increases the risk of wounding adherent veins, the more so because, as will be readily understood, in this method veins and other structures must frequently be drawn quite out of their normal position).

The finger now exploring the wound detects the position of other glands, which can be freed to a certain extent by means of the finger alone, seized and removed in the same manner; the deeper glands may be, and often are, firmly attached to the large veins of the neck, and it is therefore prudent to only partially excise these, the remaining adherent parts being scooped out as cleanly as possible with a sharp spoon; in this way almost all the gland structure can be removed, leaving, however, a part of the capsule behind.

In the foregoing manner glands can be removed quite a distance from the incision by using the fingers of both hands, one on the skin and the other exploring the wound; in the cases where more than one incision is used, glands can be pushed up by the finger in one wound to present at the other. Having completed the removal, the wounds are sutured. Except in the worst cases where there have been frequent attacks of periaadenitis, rendering the adhesions extremely firm, very large masses can be

removed, both from the anterior triangle, the posterior triangle, and from beneath the sterno-mastoid.

Secondly. In cases where the glands have broken down, unless the products of caseation have perforated the gland capsule, it is generally possible to remove the diseased glands in the same manner, care being taken to avoid puncturing the abscess; this, however, may be impossible, especially in the case of the deeper ones adherent to the vessels. In these all the caseous matter can be removed with a scoop, and as much of the capsule as possible dissected away; the wound subsequently being carefully sutured.

It not infrequently happens that tuberculous glands undergo an acute inflammation with periadenitis—especially the cases classified under type A. When there is much brawny induration and redness of the skin, with deep fluctuation, the best plan is to make a small incision deepened until pus is reached, and with a scoop to remove as much of the broken down gland as possible. This being done, in many instances the remaining inflamed glands will subside, or when the inflammatory trouble has disappeared—as it will do if the patient be kept in bed and hot fomentations applied—a clearer idea as to the necessity for excision and the scope of the operation needed can be obtained. In many cases the above trivial operation, followed by a few weeks' rest in bed, will lead to the almost complete subsidence of the swelling.

Thirdly. A case may first be seen where the glands have completely broken down, and the capsule perforated, the pus, perhaps pointing subcutaneously. Here operation is imperative to prevent destruction of the skin. If the latter be much damaged it should be included between two curved transverse incisions, the broken down material removed with a scoop, other glands if possible dissected out, and the wound sutured.

Lastly a patient may first come under treatment where the disease has existed for long periods and has been neglected, sinuses having formed. If there are palpable glands, these should be removed, the position of the incision depending on the site of the sinuses, the edges of which must be pared away. It may be possible to excise the track of the sinus with the remains of



the diseased gland to which it leads; in most cases, however, the scoop will have to be relied upon to get rid of the tuberculous material.

In many such neglected cases there will be very little gland substance left, but numerous sinuous and deep sinuses discharging pus. Here again the edges should be pared and the sinuses thoroughly scraped with a spoon, taking care to reach the deepest recesses; subsequently the surfaces should be well rubbed with wool soaked in one in twenty carbolic, carried on a probe down to these deepest recesses. Lastly, iodoform should be applied, either by means of a wool-bearing probe, or injected as iodoform emulsion. Antiseptic dressings should be applied very firmly, and left undisturbed for two or three days, the after-dressings, which should be frequent, being conducted with strict antiseptic precautions.

In all cases where the wound is closed, the greatest care should be taken to effect accurate approximation of the edges, very small needles and the finest gossamer gut being used. Dressings should be applied as firmly as is consistent with a free breathing way. The head should be supported by sand bags for a week. The stitches should be removed on the fourth or fifth day, firmly applied dressings, which lessen movement, being insisted upon for at least a fortnight, and the patient not allowed to leave his bed until four weeks have elapsed.

In those cases where tuberculous matter has been removed piecemeal with the scoop, *i.e.*, where the edges of the wound have been exposed to tubercular infection, at or about the end of the third week there may appear evidence of such infection as shown by a bluish red discolouration and the formation of nodules in the scar. Under these circumstances, the whole of the latter should be included between two elliptical incisions and excised. At the same time, if there is any tuberculous matter beneath, this should be removed by scraping and flushing. The wound should then be carefully sutured. It may, although rarely, be necessary to repeat this operation even more than once.

Again, in cases where the wound has not been sutured—where there have been pre-existing sinuses—whenever unhealthy

granulations form, these should be removed with a scoop. To ensure a good result, this slight operation, which does not necessitate the administration of a general anæsthetic, may have to be repeated many times.

The operation for excision of glands described above, appears tedious, and the admission that the removal is often incomplete would seem to afford serious objection to this method. The latter objection applies equally to erosion of joints, and I believe that in both cases these apparent drawbacks are more than outweighed by the advantages gained.

There is no doubt that the scars left from transverse incisions, even if several have to be adopted, are far less disfiguring than that from a long vertical one.

Recurrences seldom occur, unless the patient is exposed to such unfavourable conditions as those which are entailed by a sedentary life in London or large towns.

# NOTES ON THE SETTING UP AND WORKING OF AN X-RAY INSTALLATION.

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By E. W. H. SHENTON.

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THE present methods of exciting an X-ray tube—if the influence machine be neglected—demand a supply of direct current, which, however it be derived, whether from the mains or from a home installation, is the *sine qua non* of radiography. The relative merits of influence machine and coil need not here be discussed; each claims for itself advantages and each is found to have its drawbacks, but the outcome of the subject is that the coil is the most generally used instrument.

In setting up an X-ray installation the supply of electrical current available must be considered and the design should be based upon this. There are four situations in which the radiographer may find himself placed—

Firstly, where direct current is supplied to the building usually at a voltage of from 100 to 200.

Secondly, where alternating current is supplied at 100 to 200 volts.

Thirdly, where no current is supplied to the building but within a convenient distance direct current is available for charging accumulators.

Fourthly, where no electrical energy is available, and some small installation must be fitted up.

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The first condition, where there is an abundance of direct current supplied through the mains, is the most favourable. If the voltage does not exceed 100 it is possible to have an interrupter and coil adjusted to take such a current, and on this system the radiographic department of Guy's Hospital has been worked for a few months. The simplicity of this arrangement lessens the number of possible breakdowns which are so frequent when the current is supplied from storage batteries. It is not, however, the good fortune of many to be able to get a direct current at such a comparatively low voltage or even to get a direct current at all, for there are very few places in London where this is possible. Where a direct current of a voltage exceeding 100 is supplied, it will be necessary to interpose accumulators between the mains and the Röntgen apparatus. The disadvantages are chiefly in the initial expense of accumulators, a waste of current in their charging and the various evils to which accumulators are liable. The advantages are those accruing from the ease with which any voltage can be obtained. Any number of secondary cells whose sum total of volts, taken at 2.5 per cell, falls below the voltage of the mains may be safely charged by allowing the current to pass first through lamps wired up in parallel, *i.e.*, so that the more lamps used the greater will be the current. Electricity is, of course, wasted in the lighting of these lamps, but the extreme simplicity and safeness will recommend the system to the practical man. The number of cells should be decided by the required voltage. Supposing this to be twelve, and six cells are required, it would be advisable to have six, twelve or eighteen accumulators, or some multiple of six, so that they may all be charged at once and discharged in sets of six. Where accumulators are not available a very simple and efficient method of obtaining any voltage from a high constant main is to pass the current through water by means of lead electrodes—an earthenware jar and two pieces of lead pipe will serve. By altering the distance between these, or adding sulphuric acid to the water, any desired voltage may be obtained. The system, however, is wasteful and the fumes necessitate the jar being placed in the open.

The second set of conditions where an alternating current only is available, is next best. It is only within the past few months that a simple and efficient means of converting alternating current into direct current has come to light. Dr. George Batten, assisted by Mr. George Sutton, has designed a little instrument, called a rectifier, which is being manufactured by Mr. Dean, of Hatton Garden, by means of which an alternating current of any voltage is broken up into two direct currents of half that voltage or less, and is regulated by means of a transformer on the main supply. These two currents are arranged to pass along these wires, and by connecting the central wire to the middle of a set of accumulators while the two side wires are attached to either end, all the current is utilized in the charging process. The

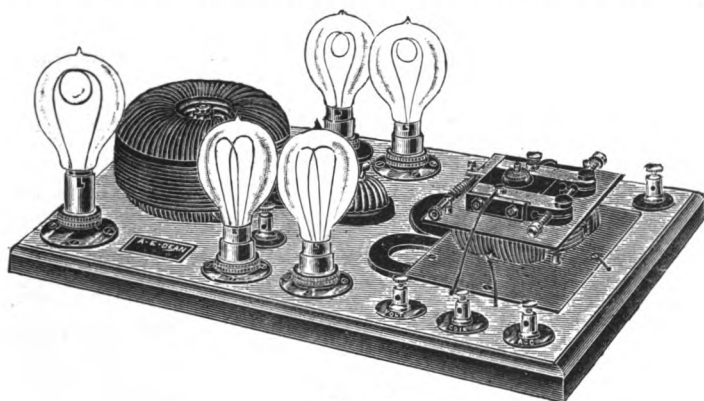


Fig. 1.

block (Fig. 1) will show the nature of the instrument. It can be used to work a coil direct without the interposition of accumulators by using the current derived from it through an electrolytic, or even a platinum hammer brake. This little piece of apparatus will be of the utmost value to anyone desiring to charge accumulators from an alternating current, both on account of its low price and the absence of complicated mechanism. Through the kindness of Dr. Batten I have been able to test the instrument and can speak with confidence of its practical worth. The diagrams here shown will serve to explain the principle. There are methods of utilizing an alternating current for producing the X-rays without

transforming it, but these can scarcely be said to be satisfactory, and coils and tubes can easily be spoilt in experimenting with a multipolar current.

Little need be said of the third set of conditions except that they are suitable only where the work is light.

Where no electrical energy is available as in the fourth set of conditions a small installation must be fitted up. This should consist of a gas or oil engine, dynamo, and set of accumulators. The engine should not be less than one-horse power nor the dynamo than half a unit. The voltage of the dynamo will depend upon the number of accumulators and these again upon the voltage decided upon. Under these conditions I believe 25 volts to be a very efficient current, and this would necessitate twelve cells in the storage battery, and a shunt wound dynamo with an output of 30 volts and with an ampérage dependent upon the size of the accumulator cells.

Supposing that a direct current of a steady voltage has been secured, the Röntgen apparatus is the next consideration.

The interrupter is so vital a part of an X-ray apparatus as to need special mention, and will therefore be discussed first.

The purchaser of apparatus will, as a rule, be most careful in the selection of his coil, while to the interrupter he will scarcely give a second thought, as being of very secondary importance. In this he is mistaken. Three classes of interrupters will present themselves; the platinum, the mercury, and the Wehnelt; and there is much for and against each.

The platinum has the virtue of simplicity, but of nothing else in particular.

The mercury break has the disadvantage of being mercurial and therefore somewhat messy if not well treated, but its virtues are numerous. It is the most economical where current is concerned. The speed of interruption is governable and has wide limits. The effect upon the tube is such that the very maximum of Röntgen light can be obtained, a result brought about possibly by the condenser current, which is made more use of in this form of break than in any other. When once started it runs continuously, and the apparatus can be safely left, as at times is

very desirable, for instance, while walking to the other side of the couch during screening.

The Wehnelt has many admirers, and its effect upon the Röntgen light is certainly wonderful. In place of the slight flicker discernible in even the best form of mercurial break a steady light of considerable intensity is obtained. But the disadvantages of the Wehnelt are many. It fumes badly, it is noisy, and it takes much more current than any other form of break. It is liable to damage tubes from the heating effect produced at the anode, and finally where the very best is required from a tube it fails owing, I believe, to the loss of condenser discharge, the result of the rapid interruptions.

The choice, then, of an interrupter is simplified by the consideration of the above facts. The mercurial in some form will be wisely adopted by those who desire a trustworthy and workmanlike instrument. Of mercurial interrupters there are many forms, but the qualities which good work will demand are, firstly, that the motor mechanism shall be well made in a heavy pattern so that steady running may result. Fig. 2 shows the type of interrupter used in this department and its working may be well understood after a careful survey. The small steadily-running electro-motor actuates a crank which raises or lowers a copper rod in a glass vessel. This vessel holds a small one containing the mercury and in and out of this mercury dips the copper rod when the motor is running. The glass vessel can be raised by a screw so that the rod will dip a long way in the mercury and come out of it but a short distance, or the opposite state of affairs may be brought about by reversing the screw. In other words, the "make" can be increased at the expense of the "break" or *vice versa*. The outcome of this adjustment is seen in the excited tube where the length of "make" will be apparent by an increased volume of light and possibly by the heating of the anode. The relation of "make" to "break" is decided upon by the degree of resistance offered by the tube—a low or soft tube requiring less "make" than a high or hard tube.

The mercury is covered with methylated spirit contained in the outer glass vessel. The spirit washes away the oxide formed at the surface of the mercury by the spark of the interrupted current. It should be mentioned that the motor itself is run on a shunt current, and this modified by resistance enables any speed to be obtained independently of the quantity of current used in the coil.

The crucial test of a mercurial break is not how quickly it will interrupt but rather its adjustability from too fast to too slow. It is perhaps unnecessary to state that it is convenient to have a motor wound to take the same voltage as the coil. They are made with windings to suit from 12 to 100 volts. It will not be necessary to say very much about coils. There are many very excellent makes, and if they will fulfil the following requirements they will suit.

(a) A spark of ten inches produced at each interruption, of a thick, furry nature. (The thickness of the spark may be somewhat increased by lengthening the "make," as described above.)

(b) It should only take a reasonable ampèreage to produce this, say not more than 5 amperes at 12 volts.

Among details in construction, section winding and the extension of the primary at either end, seem to add materially to efficiency.

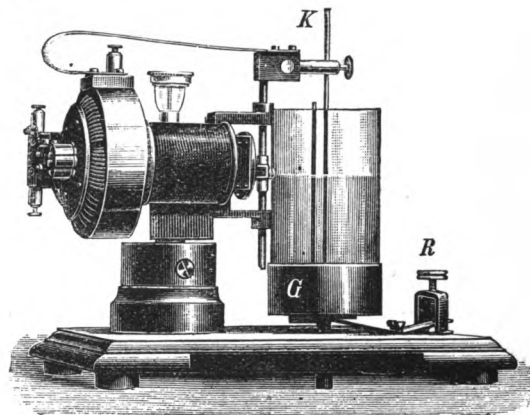


Fig. 2.



The two important items, viz., interrupter and coil being disposed of, it remains to show a suitable way of setting up and arranging the apparatus.

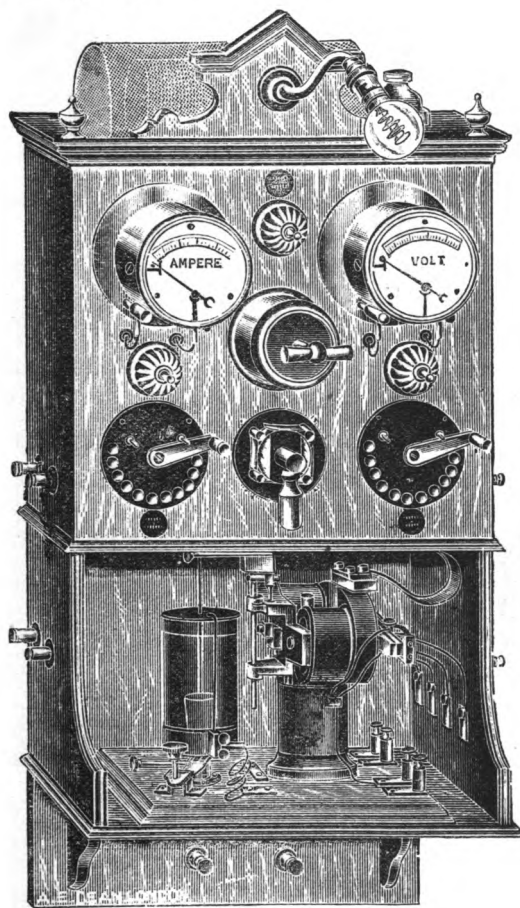


Fig. 3.

The interrupter is best fitted to a switch-board hung upon the wall (Fig. 3). The board should contain a switch to start the motor and a switch to put the current into the coil. It is as well to have these two combined in one, so that at the first turn the interrupter starts, at the second the coil. This prevents any possible chance

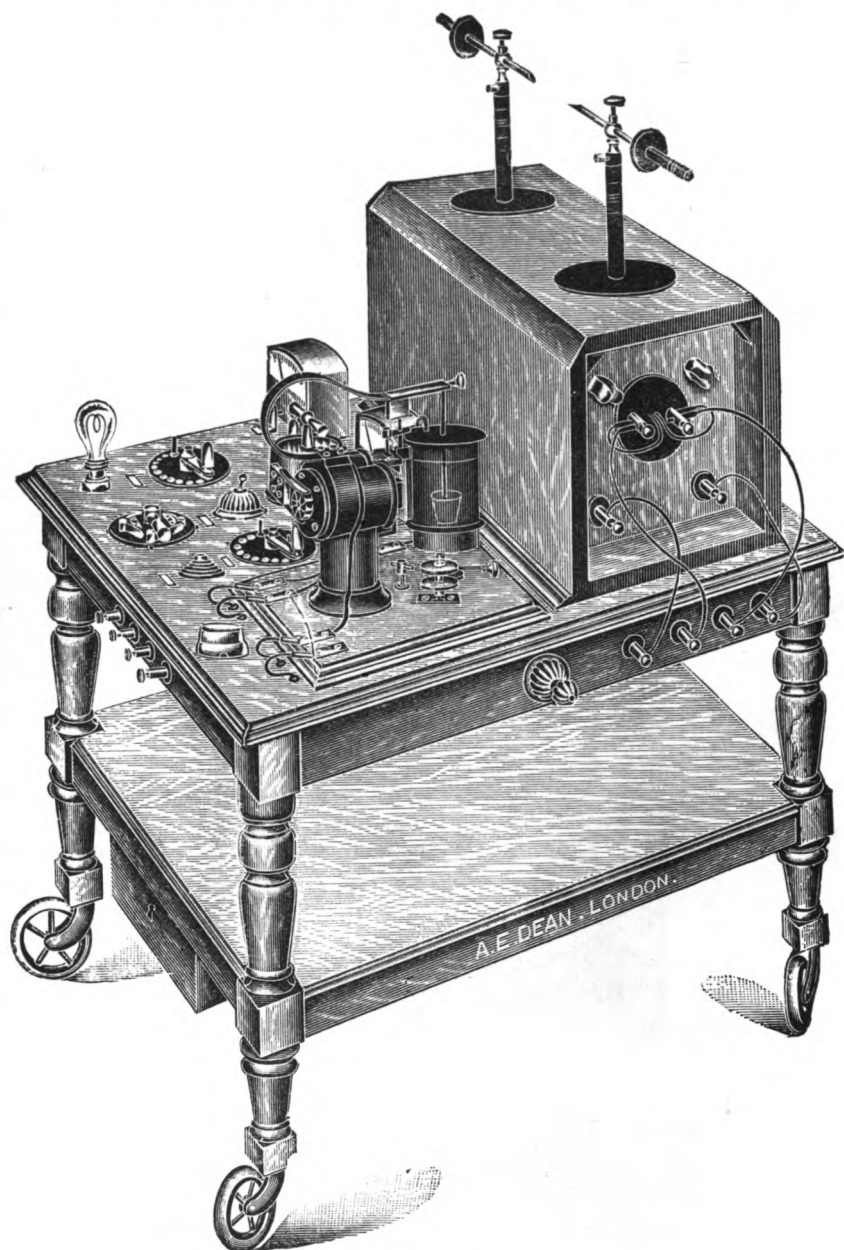


Fig. 4.

of the main current being turned on while the motor is not running. There should be two regulating resistances—one in the interrupter circuit, one in the coil circuit, to respectively modify the rate of interruption and the quantity of current in the primary of the coil. It is convenient to also have on this board a voltmeter and an ampèremeter. The first on a shunt circuit from the battery, the second in the primary circuit. Both should be capable of being cut in and out by switches.

If the room switch for the electric light is not near by, it is advisable to have a lamp upon the board, worked from the batteries. Fig. 4 shows the same switch-board in a portable form.

The next consideration is the position of the coil. It should be placed either high up on a shelf about six feet from the ground or quite low, *i.e.*, from twelve to eighteen inches from the floor. In either case it should not be far from the switch-board.

The couch upon which the patient is to be examined must next be considered. Here the operator has to decide whether he will have his tube below and the screen above the patient, and work in comfort, knowing what he is photographing, or whether he will have his tube above and push his plate beneath, and trust to an ever failing luck to get the view he requires. There can be no question as to which method is theoretically and practically best; but many radiographers still work with the tube above.

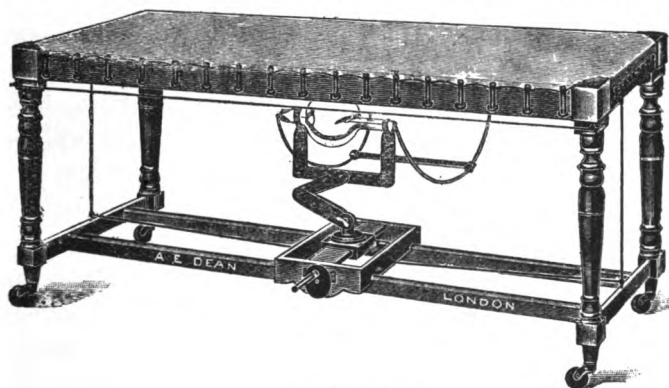


Fig. 5.

The couch (Fig. 5) which Mr. Dean has made to my design, and which is in use in this and several other hospitals, consists

of a table the top of which is canvas, upon which the patient lies. Beneath the table is the tube holder running to and fro upon guides and from side to side by a screw mechanism. The movements of the tube can be guided by the operator as he looks at the screen. This latter is placed upon the patient. When the view on the screen is found to be satisfactory, a plate, in the usual light-tight envelope, is pushed beneath the screen, and the tube once again illuminated. It is quite possible now to see the plate as well as the image being photographed, and to know whether this latter is well placed. It is difficult to believe how greatly this couch facilitates the work, or how it adds to the comfort of both operator and patient.

The most important piece of apparatus—the tube—has been left until last. Without a good tube there cannot be good work. It is upon this fragile glass thing that the radiographer depends for his success, and the surgeon for the helpful facts which may clinch a diagnosis, and a chance knock may shatter the hopes of both. It is not only the expense of a tube that has to be considered in such an unlucky accident, but the impossibility of replacing it with one of equal value, for there is a lot of work to be done to a tube before it is in good working condition. Tubes differ in disposition and usefulness almost as much as do human beings. It appears a nice theory to have a battery of say a dozen tubes, and use each in rotation so that the others may be resting, but in practice this is found not to be practicable. One tube will at once assert its superiority, and consequently be used where the maximum of light is required, and this will be in almost every case. The remaining eleven will therefore be left untouched, and the best tube is kept upon the holder until over-work or ill luck destroys it.

The tube (Fig. 6) used in the Guy's Hospital Radiographic Department is one that has been made from my suggestions by Mr. Dean. It is extremely large, measuring over six inches in the diameter of its bulb, and has two instead of three electrodes. With this tube it is possible under favourable conditions to get skiagrams of the stoutest individual in any region, in a space of

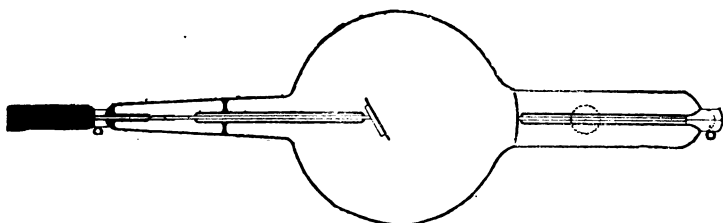


Fig. 6.

time that need not exceed half a minute. Its screening capacity is great, and upon many occasions I have been able to see renal calculi *in situ*.

Where tubes of high illuminating quality are in use it is possible to do any localisation of foreign bodies that is to be met with in radiography except those in the eyeball, for very often these are so small as to be incapable of demonstration upon the screen.

It will be observed that as far as possible all the apparatus here described is of a simple nature, and free from the elaborations and complications that hamper the operator where they pretend to help him. And yet with such an outfit the very best and most satisfactory work may be done. I have stated before that exposure need never exceed thirty seconds, but it is not often that so long an exposure as this has to be given; hands, feet, and other transparent regions can be recorded in a mere flash; elbows, knees, and other portions of the limbs in three or four seconds. Some of the best skiagrams of renal and bladder calculus taken in this department have been obtained in an exposure of from ten to twenty seconds.

Having referred to the possibility of localising upon the screen without the aid of special apparatus, it will be as well to describe this method briefly. Take as an example the localisation of a bullet in the thigh. The patient is brought into the room, probably upon a stretcher, and to avoid unnecessary movement the stretcher may be placed at once upon the couch. The extra thickness of canvas will not materially obstruct the Röntgen rays. The room being now darkened, the current is turned on and the tube illuminated. The screen is placed upon the injured thigh, and the operator moves the tube by the leather band at the side of the

couch until the bullet is seen clearly. At this stage he takes a probe and places it by the side of the limb, at a spot as near to the bullet as he can select. Both bullet and probe being well shown upon the screen the operator or his assistant moves the tube from side to side so that the shadows upon the screen oscillate. If the probe is not lying upon the same plane as the bullet the shadows of each are seen to move at different rates, that which is nearer the screen surface moving slower than that which is more remote. The operator now has only to adjust this probe so that its shadow and that of the bullet move at the same rate, or, what is equivalent, the same distance, and he knows that both are upon the same plane. In other words, his probe is exactly the same distance from the surface of the screen as the bullet. The small instrument depicted in (Fig. 7) is a substi-

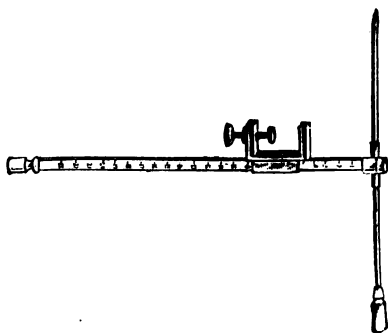


Fig. 7.

tute for a probe, and exactly records the distance, of the object sought, from the screen. This system (which I have exhibited at the Clinical Society of London) with various modifications, has been used for several years at Guy's Hospital, and has replaced all others where apparatus was necessary. It does not involve any photographic process and is perfectly accurate.

The examination of the head, neck and thorax do not need special remark, the patient being placed upon the couch either upon his back or upon his face. For the investigation of the abdomen the patient should lie upon his face with his arms above his head so that he is resting his chest and abdomen

on the canvas. If it is found that the trunk is not sufficiently transparent it will be as well to insert a pillow under the abdomen, which will tend to lessen the antero-posterior diameter. In this position, while the tube is moved upwards and downwards the length of the couch, a careful watch must be kept upon the screen to note abnormalities. Calculi will appear as dark masses generally showing a little below the costal margin, as this is the position in which they are most evident upon the screen. Foreign bodies will arrest the eye by their unusual appearance.

In cases of doubt as to the penetrating power of the tube it is well to push a coin or small metallic object beneath the patient and to notice whether it is visible upon the screen. It should be quite possible to see such an object, even through the head. All four limbs can be examined separately by getting the patient to move a little over toward the further side of the couch and allowing the limb to come well down upon the canvas above the tube. If the very maximum of light is required the tube can be brought quite close up to the canvas without fear of a chance movement of the patient bringing him too near. In examining a knee in the lateral position the patient should lie upon the same side as the affected joint and bring the other leg across in front of the one examined; this will ensure a much better view than any other arrangement. The tube and patient should be so arranged that the patella is seen standing clear of the other bones. One of the most trying regions to screen and photograph satisfactorily is the ankle, looked at antero-posteriorly. To do this it is as well to extend the foot as much as possible.

It is often desirable to set a fracture while the bones are being watched upon the fluorescent screen. It must not be supposed that, by this means, accurate positions are obtained, for one of the most striking features of such treatment is how very little extension and other forms of manipulation affect the arrangement of broken bones. However, it cannot be denied that it is a great help to good results if the process of putting up can be viewed. Some arrangement will be necessary where an anæsthetic is being administered that light may be turned up immediately when

required. The tube must be firmly settled in its holder so that the shaking of the couch may not upset it.

The usual type of tube holder only grips the tube at one end and the leverage is therefore very great. The kind which Mr. Dean makes from my design supports the tube at both ends and lessens the danger of breakage.

The apparatus here detailed will be found ample for the requirements of an X-ray clinique. Success will only come by practice, and no amount of accessory will serve as a high road thereto. It is unfortunate that many enthusiasts seem to degenerate into collectors of useless and obsolete apparatus, when their misspent energy would have made them expert radiographers had they kept to simple tools.



FUNCTIONAL PULMONARY  
INCOMPETENCE, AND DILATATION AND  
ATHEROMA OF THE PULMONARY  
ARTERIES, AS COMPLICATIONS OF  
MITRAL STENOSIS.

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By J. H. BRYANT, M.D.

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THE occurrence of pulmonary regurgitation as a complication of mitral stenosis has interested me for some time, and I have in consequence carefully examined, from this point of view, all the cases of mitral stenosis I have seen. During the six years I was holding the post of Medical Registrar I had exceptional opportunities for carrying out these observations, having had access to all the cases of this nature admitted to the medical wards, and I take this opportunity of thanking my colleagues for allowing me to make use of the notes of them. I have collected sixteen cases bearing on this point, the largest collection I believe hitherto published. My interest in this condition was first aroused by Case 1. When she was admitted on the second occasion, in addition to a rumbling presystolic bruit at the point of the cardiac impulse there was "two inches to the inner side of the left nipple and traceable upwards into the third left space,

a whistling early diastolic bruit." Three explanations were suggested to explain the occurrence of this bruit—

- (1) That it was due to the mitral stenosis.
- (2) That it was due to aortic regurgitation.
- (3) That it was due to pulmonary regurgitation.

I must confess that I first of all favoured one of the first two views. Dr. Goodhart subsequently saw the case, confirmed the presence, character, and position of the bruit, and definitely stated that in his opinion the murmur was due to pulmonary regurgitation, the result of a dilated pulmonary artery. This opinion was verified by the post-mortem examination which I performed on March 14th, 1893. The pulmonary artery was decidedly thickened and dilated, it measured 101·6 millimetres in circumference, the average circumference of the pulmonary artery in females being about 88 millimetres.

Adams recorded two cases of mitral stenosis in which dilatation of the pulmonary artery was found. In the first case it is thus described: "The pulmonary artery was unusually dilated." In the second case: "The pulmonary trunk was dilated nearly to twice its natural size." These are two of the earliest cases I have been able to find recorded.

Stokes describes a case of dilatation of all the cavities of the heart and of the pulmonary artery and the aorta, but makes no mention of pulmonary regurgitation from dilatation of the pulmonary artery, as a complication of mitral stenosis.

To Graham Steele belongs the chief credit for drawing attention to this form of pulmonary incompetence. In his work entitled, "The Physical Signs of Cardiac Disease," when writing of the regurgitant murmur resulting from dilatation of the aorta, he says, "I am inclined to believe that a murmur of similar mechanism occurs on the right side of the heart when there is much obstruction to the pulmonary circulation with a dilated pulmonary artery." The same physician in 1889, in an article entitled, "The Murmur of High Pressure in the Pulmonary Artery," writes, "I wish to plead for the admission among the auscultatory signs of disease of a murmur due to pulmonary regurgitation, such regurgitation occurring independently of disease or deformity of the valves, and

as the result of long continued excess of blood-pressure in the pulmonary artery." He points out that in cases of mitral stenosis an early, soft blowing, diastolic bruit following or immediately running off from an accentuated pulmonary second sound, may be heard at the sternal ends of the third and fourth intercostal spaces. If the second sound happens to be reduplicated the murmur usually follows the latter sound. This bruit is not usually persistent at first and shows great variableness in its intensity, but the accentuation of the second sound is always present.

Barr, when writing of the bruit associated with mitral stenosis, says, "The early diastolic murmur may be purely mitral in origin, but I am convinced that it is often confounded with a short, soft blowing, diastolic murmur, which not infrequently occurs in this disease at the moment of closure of the pulmonary valve, and which arises from slight regurgitation into the right ventricle owing to the high pulmonic tension." And later, writing of the pulmonic second sound he says, "In a certain number of cases there is a short, soft, diastolic murmur (to which I have before alluded) in the pulmonic area, due to slight regurgitation through the pulmonic orifice. This is especially apt to happen when the right ventricle is dilated and does not completely empty itself during systole, the high tension in the pulmonic system drives a certain amount of blood back through its closing valves into the unemptied ventricle. Some think this impossible, as the valve is usually found competent at the autopsy, but there is a remarkable difference between the pressure of one or two inches of water and the pulmonic tension in a case of mitral stenosis."

Sansom says, the early diastolic murmur associated with mitral stenosis when there is no other sign except the murmur, is indicative of aortic regurgitation, and he criticises Graham Steele's explanation that they are due to regurgitation into the right ventricle in consequence of the extreme tension within the pulmonary artery caused by the mitral obstruction. He quotes Percy Kidd, who objects to this view of Graham Steele's on the grounds that if the murmur were really due to this cause there would be some evidence of leakage or valvular defect found not infrequently at the necropsies made on cases of mitral stenosis. Sansom thus

writes, "I am not aware of a single case that has demonstrated either structural defect or passive dilatation permitting regurgitation through the valves of the pulmonary artery, and I, therefore, regard the theory as unproven. It is also in my opinion improbable, seeing that such diastolic murmur is, so far my experience goes, unknown in other conditions—very grave mitral regurgitation for instance—when the pulmonary artery is subject to severe internal pressure. Moreover, if it were true, I think we ought to be able to trace in dilatation of the pulmonary artery the replacement of the loud pulmonic sound heard in cases of mitral stenosis and mitral regurgitation by a diastolic murmur, or the affixing of such a murmur to the exaggerated normal sound. I have never met with, nor have I heard of any such experience." He considers the diastolic murmur to be due to either the mitral lesion itself or to aortic regurgitation. I do not consider that these arguments are conclusive, for the pulmonary artery is a highly elastic structure, and it is quite reasonable to suppose that as soon as the patient dies and the increased pulmonary tension is relaxed, the dilated pulmonary artery, if moderately dilated, contracts down to its normal size. One of the chief characteristics of the bruit is its variability, and this may be explained by a disappearance of the dilatation under favourable conditions. Case 16 illustrates this point, for the bruit was heard on March 17th and 20th, but not on the 23rd or subsequent dates. There is, however, conclusive evidence to show that in some cases of mitral stenosis there is appreciable dilatation of the pulmonary artery.

Duckworth has reported a case of tricuspid and mitral stenosis in which symptoms of pulmonary incompetence were present. The patient was a married woman, æt. 23. A diastolic murmur was heard in the pulmonary area but subsequently disappeared. At the post-mortem examination the pulmonary valves were not markedly altered. The mitral valve was much stenosed and button-hole in shape. There was decided dilatation of the pulmonary artery. "The pulmonary arterial reflux was probably explicable by the dilated state of the vessel and the disappearance of the murmur was coincident with increasing low blood-pressure, as the vital powers failed towards the last."

I have divided my cases into two groups, the first nine in which post-mortem examinations were made, the last seven in which the diagnosis of the condition rested on the clinical evidence alone.

The post-mortem evidence of dilatation of the pulmonary artery, then, is as follows :—

Case 1. The pulmonary artery appeared to be dilated, and it felt much thicker than normal, almost as thick as the aorta (Drs. Goodhart and Shaw confirmed this). The pulmonary orifice measured 91·5 millimetres, and 20 millimetres above the orifice the circumference of the vessel was 101·6 millimetres. In addition to this I noted that the pulmonary valves appeared to be very thin and loose, and each corpus arantii was considerably thickened. The free edges of these valves were slightly curled towards the ventricle. There were no recent vegetations, and the valves were thin and loose. It was evident that these valves had not been involved by any previous attack of endocarditis. The measurements showed considerable dilatation, not only of the pulmonary artery but of the orifice itself.

Case 2. The pulmonary artery appeared to be much larger than normal, for it seemed almost double the size of the aorta. The pulmonary orifice measured 8 centimetres and 12 millimetres above the orifice the circumference of the lumen was 10·16 centimetres. Although the measurements do not point to much increase in size, the appearance of the vessel at the time of the post-mortem certainly indicated a considerable amount of dilatation, and allowing for a certain amount of contraction after death, when the tension had relaxed, it would seem that during life there must have been considerable dilatation.

Case 3. The pulmonary artery was thickened and dilated; it was almost as thick as the aorta; 2·5 centimetres above the pulmonary valves it measured 8·25 centimetres in circumference when laid open.

Case 4. In this case there was undoubted post-mortem evidence of dilatation of the pulmonary artery. Dr. Perry described the condition as follows: "On the right side the pulmonary valves

measured 11·4 centimetres in circumference, and, therefore, there was doubtless pulmonary regurgitation."

Case 5. The pulmonary orifice measured 7 centimetres, just above the orifice the artery measured 8·8 centimetres, and had the appearance of being considerably dilated and was much larger than the aorta. In this case again the actual measurements did not show much dilatation, but the appearance of the pulmonary artery and its size when compared to the aorta certainly pointed to an abnormal distension of that vessel.

Case 6. The pulmonary orifice measured 7·6 centimetres. The pulmonary artery was dilated, and was much larger than the aorta.

Case 7. The pulmonary orifice measured 7·6 centimetres. The pulmonary artery was dilated and was much larger than the aorta.

In cases 6 and 7, therefore, although the measurements did not show actual dilatation, a comparison with the aorta showed that the pulmonary artery in each case was undoubtedly abnormally large.

Case 8. The pulmonary artery was much larger than the aorta, was much thicker, and had atheromatous patches in it. It measured 7·6 centimetres across, against 5·7 centimetres of the aorta. In this case also, although the measurement was not great, there was undoubted dilatation of the pulmonary artery.

Case 9. The pulmonary artery looked very large, the valves measured 8·9 centimetres, and were normal in appearance.

In cases 10, 11, 12, 13, 14, 15 and 16 the evidence of pulmonary incompetence from dilatation of the pulmonary artery rested on clinical evidence alone. Very strong evidence can be adduced of increased tension in the pulmonary arteries in cases of mitral stenosis by post-mortem examination. I have never performed or seen performed a post-mortem examination on a case of advanced mitral stenosis without finding thickening, dilatation and atheroma of the branches of the pulmonary arteries in the lungs. Very little attention, if any, is drawn to this change in the branches of the pulmonary arteries in the descriptions given in the various text-books, of the backward pressure effects of chronic

monary Arteries.

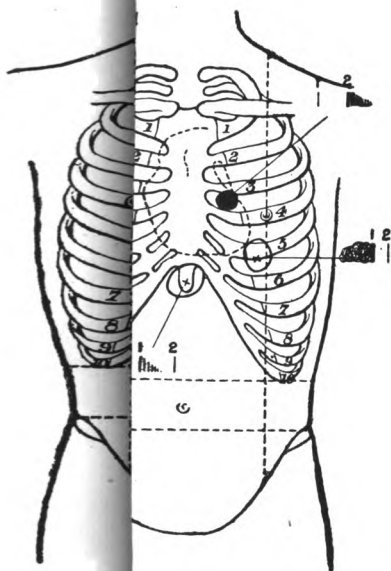


Fig. 4.

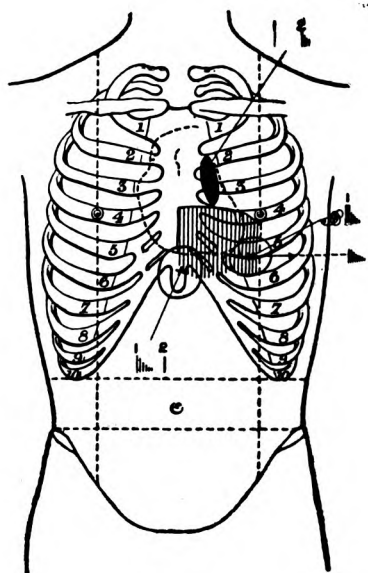


Fig. 5.

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valvular disease of the left side of the heart. I claim that these changes in the pulmonary arteries should find an important place in all descriptions of the effects of backward pressure on the other organs of the body from chronic valvular disease of the heart.

Hilton Fagge drew attention to this point. He wrote: "The increase of tension in the pulmonary vessels soon leads to changes in their walls, which become thickened and hypertrophied. In the main trunks of the pulmonary artery this is particularly noticeable. The records of post-mortem examinations at Guy's Hospital contain notes by Dr. Moxon of the case of a boy, aged ten years, in whom the coats of the pulmonary artery were nearly twice as thick as those of the aorta at its thickest part, and less striking examples of the same are very commonly met with. The artery also becomes greatly dilated." Fagge also states that another important result of this increased tension is an atheromatous change in the branches of this vessel, and he quotes a striking example of this condition, recorded by Dr. Conway Evans.

It appears that the first observer to call attention to these changes in the pulmonary arteries was Dittrich, who described it as occurring particularly in the smaller branches, and looked upon this change as being the actual cause of the pulmonary apoplexies which are so frequently met with under such conditions.

Yeo has published a case of atheroma and dilatation of the pulmonary arteries secondary to mitral stenosis and aortic disease. "The pulmonary artery from the semilunar valves down to the very first subdivision is irregularly dilated and inelastic, the inner coat throughout being studded with hard prominent yellow patches, some of which are rough on the surface and look like ordinary atheromatous ulcers." The mitral valve was much stenosed. He considered the change to be due to the increased tension and consequent mechanical irritation.

Pepper also reports a case of sclerosis and atheroma of the pulmonary arteries secondary to mitral stenosis.

Whittaker writes: "Sometimes the valves are perfectly sound, but the orifice is dilated, so that the condition is that of relative insufficiency. The cause in these cases is usually an arterio-

sclerosis which produces dilatation of the pulmonary artery and the cause of the arterio-sclerosis is for the most part syphilis."

I quite agree that the valves may be perfectly sound and that the orifice may be dilated and so produce a relative insufficiency. I also agree with the fact that arterio-sclerosis is usually associated with this condition, but I do not consider there is sufficient evidence to show that syphilis has any direct connection with it. There is, however, conclusive evidence that increased tension is the most important causative factor, for I maintain that it is more frequently observed as a complication of mitral stenosis than of any other condition, but it is also associated with other chronic valvular lesions of the left side of the heart and with pulmonary emphysema. I have only seen one case in which no obvious cause was found which could have given rise to increased pulmonary tension.

Mott, when writing on the important influence of internal strain as a factor in the production of arterio-sclerosis says: "The relative infrequency of affection of the pulmonary artery which occurs, indeed, only under such conditions as involve increased tension of its walls, as, for example, prolonged mitral stenosis, indicates the importance of internal strain as a factor in the degenerative process."

Osler writes: "Sclerosis of the pulmonary artery is met with in all conditions which for a long time increase the tension in the lesser circulation, particularly in mitral valve disease and emphysema."

Sometimes the sclerosis reaches a high grade and is accompanied with aneurysmal dilatation of the primary and secondary branches, more rarely with insufficiency of the pulmonary valve."

The description of the condition of the pulmonary arteries in the cases in which post-mortem examinations were made is as follows:—

Case 1. The branches of the pulmonary arteries in the lungs were atheromatous, more especially on the right side and in the right lower lobe.

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Case 2. The pulmonary arteries were thickened, dilated and atheromatous, and stood out prominently from the cut surface of the lungs, with open lumina.

Case 3. The two main branches of the pulmonary artery were thickened and atheromatous, and the small branches in the lungs were thickened, atheromatous, and dilated, and stood out prominently from the cut surface of the lung, with open lumina.

Case 4. In the pulmonary artery were atheromatous patches, and in the left branch of it was a small piece of ante-mortem clot.

Case 5. The branches of the pulmonary arteries in the lungs were thickened and atheromatous, standing out prominently, with open lumina from the cut surface of the lung.

Case 6. On section (of the lungs) the pulmonary arteries stood out very prominently from the cut surface; they were dilated, very much thicker than normal, showing marked atheromatous changes, and appearing in fact almost as thick as similar sized bronchial tubes.

Case 7. The intima of the pulmonary artery was thickened and the larger branches showed marked atheromatous changes.

Case 8. The pulmonary artery had atheromatous patches in it.

Case 9. The pulmonary branches were greatly dilated and the coat showed irregular cracks in the intima, which were blood-stained. These appeared to be due to over distension; there was no atheroma.

In all the cases, therefore, in which a post-mortem examination was made there was very strong and conclusive evidence of strain and increased tension in the pulmonary arteries. Dilatation, thickening and atheromatous patches were the changes most frequently noticed. In none of the cases was there any evidence of syphilis.

Hunter was the first to point out that the pulmonary sigmoid valves do not "do their duty" so well as the aortic valves. He proves this by experimentally injecting the arteries towards the ventricle. Gibson has made some interesting experiments on the pulmonary valves of the dead heart with a column of fluid consisting of a solution of sodium chloride of specific gravity 1050 at 98.5 F., and to quote his words the following is the

average result of his series of experiments: "From the semi-lunar valves of the pulmonary artery of the sheep a strong jet escaped until the column of fluid measured fourteen and a half inches from which height it trickled until the valves became competent with a column of nine inches. In the ox, a strong jet was emitted down to twelve inches and dropping of the fluid reduced the superincumbent column to the height of six inches, when competence was established. In the healthy human heart a jet escaped down to thirteen inches, and the valves were competent with eight inches of fluid resting on them. Now, in each case of the pulmonary valves with a column of fluid six feet in height perfect competence was obtained in a very simple manner by constricting the pulmonary artery. A cord tied round the artery exactly at the attachment of the valves gave the means of perfect control over the escape, so that by varying the amount of tightening, the jet was converted into a drop falling quickly or slowly and this in turn was totally stopped. The whole diminution of circumference amounted to a few lines. This shows clearly that the escape is caused by distension of the elastic artery and relative incompetence of the valves." It was found that the aortic valves treated in a similar manner allowed of no escape, and the experiments showed that the pulmonary orifice is not closed so perfectly as the aortic, and point to the fact that any abnormal pressure in the pulmonary artery may give rise to reflux of blood into the right ventricle.

The marked structural changes found in the pulmonary arteries may therefore be looked upon as the result of the increased tension produced in the pulmonary circulation by the obstruction to the flow of blood through the mitral orifice. Mitral stenosis produces hypertrophy and dilatation of the left auricle, and hypertrophy and dilatation of the right ventricle. It would be difficult to imagine, therefore, that the pulmonary artery, which is situated between these two hypertrophied and dilated structures, would escape being damaged. That it does not escape is shown by the changes reported in the accounts of the post-mortem examinations in the first nine cases. It is a well known fact that when there is dilatation of the first part of the aorta, a reflux of blood occurs

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into the left ventricle, and this may happen without there being any pathological change in the valves themselves. It has been shown by experiment that the pulmonary orifice is less resistant than the aorta, so that it is not difficult to imagine that the pulmonary artery and its orifice may become dilated as a result of the enormously increased tension produced by mitral stenosis, and may give rise to a functional incompetence without any actual disease of the valves.

There is also post-mortem evidence of this dilatation and incompetence, and I refer in particular to cases 1 and 4. I again repeat that the absence of post-mortem dilatation is not a conclusive proof that there was no dilatation during life. The elasticity of the artery, as soon as the increased tension was relaxed, unless the vessel happened to be enormously and permanently stretched, would assert itself, and the vessel would reassume its normal size.

Newton Pitt states that pulmonary incompetence is the rarest of all the valvular lesions of the heart, seventeen cases only were noticed during a period of twenty-three years in 11,000 necropsies. He gives a list of causes based on an analysis of 115 cases, and mentions that of this number twelve were due to dilated pulmonary artery.

In nineteen out of twenty-one cases of dilated pulmonary artery mitral stenosis was the cause. Pitt definitely concludes that there is both clinical and pathological evidence that the pulmonary arteries may become dilated in advanced mitral stenosis. He also states that in nearly all the cases post-mortem evidence of any structural change at the orifice will be absent.

#### THE CONDITION OF THE MITRAL VALVE, AORTIC VALVES, RIGHT VENTRICLE AND TRICUSPID VALVES.

In all the cases in which post-mortem examinations were made the mitral valve was much stenosed and changes were also noted in the left auricle and right ventricle.

Case 1. The mitral orifice was very small, only just admitting the tip of the little finger; it was funnel shaped. The valve was much thickened and it measured 23·8 millimetres. The chordæ

tendinæ were shortened and thickened. The aortic valves were healthy. The left auricle was hypertrophied. The right ventricle was hypertrophied and its wall measured 13 millimetres in thickness. The tricuspid valves were healthy.

CASE 2. The heart weighed 397 grammes. The mitral valve was very thickened and calcareous, and the orifice would only just admit the tip of an index finger, it measured 3·81 centimetres in circumference; it was button-hole in shape. The chordæ tendinæ were thickened and shortened. The aortic valves were normal. The left auricle was hypertrophied and dilated, and there was an ante-mortem thrombus in the appendix of the left auricle. The right ventricle and auricle were dilated, and the wall of the right ventricle was hypertrophied. The tricuspid orifice measured 10·16 centimetres.

CASE 3. The heart weighed 350 grammes. The mitral valve was thickened and calcareous, and its circumference measured 3·17 centimetres. There were a few recent vegetations on the auricular edge of this valve. The chordæ tendinæ were short, thickened, and adherent to each other. The aortic valves were healthy. The left auricle was hypertrophied and dilated. The right ventricle was hypertrophied, its wall measuring 9·5 millimetres in thickness. The right auricle was a little dilated. The tricuspid orifice measured 10·4 centimetres.

CASE 4. The heart weighed 482 grammes. The mitral orifice was much stenosed and would just admit the index finger. The aortic valves were normal. The aortic orifice measured 6·35 centimetres. The tricuspid orifice measured 15 centimetres. There were ante-mortem thrombi in the appendices of both auricles.

CASE 5. The mitral valve was thickened and calcareous, its orifice was much narrowed and measured 5 centimetres in circumference. The chordæ tendinæ were thickened and shortened. The aortic valves were thickened and adherent but appeared to be competent. The left auricle was hypertrophied and dilated. The right ventricle measured 11·45 centimetres across at the base, and its wall was hypertrophied. There was ante-mortem thrombosis of the appendix of the left auricle.

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Case 6. The heart weighed 510 grammes. The mitral valve was hard and thickened, and the mitral orifice would only just admit the tip of the little finger; it measured 37 millimetres in circumference. The chordæ tendinæ were shortened and thickened. The aortic valves were healthy. The left auricle was hypertrophied. The right ventricle was much hypertrophied, and measured two centimetres in thickness. The right auricle was hypertrophied and dilated.

Case 7. The heart weighed 510 grammes. The mitral orifice was much narrowed and thickened and was button-hole in shape. The orifice would only just admit the tip of the little finger. The chordæ tendinæ were shortened and thickened. The aortic orifice measured 5·6 centimetres; the valves were certainly thickened but were quite competent. The left auricle was considerably dilated and hypertrophied, and the endocardium was much thickened. The right ventricle and auricle were hypertrophied and dilated. There was ante-mortem thrombosis of the appendix of the right auricle. The tricuspid valve was shortened and thickened and its circumference was 12·7 centimetres.

Case 8. The heart weighed 397 grammes. The mitral valve was converted into a calcareous ring, just admitting the end of the index finger; there were a few vegetations on the edge. The edges of the aortic valve were thickened and rolled. It seemed as if there must have been some regurgitation, but no murmur was ever heard, and the left ventricle was not much enlarged. The right ventricle and auricle were dilated and hypertrophied. The tricuspid valve had thickened edges and allowed regurgitation.

Case 9. The mitral valve was stenosed; It measured 7·6 centimetres. The aorta was dilated, and the valves were thickened and doubtfully competent. The right ventricle was much hypertrophied. The tricuspid valve was thickened, and it measured 14·3 millimetres.

An analysis of the above shows that the weight of the heart was noted in six of the cases, the lowest weight recorded was 350 grammes and the highest 510 grammes, so that considerable enlargement was common.

The mitral valve was small in all the cases; the smallest measurement was 23·8 millimetres and the greatest 76 millimetres. It was thickened in seven of the cases and calcareous in four, so that it was evident that the changes were of old standing.

In five of the cases the aortic valves were described as being healthy or normal. In two, they were described as thickened but competent, and in two as thickened and doubtfully competent, but in both of these last mentioned cases there was no clinical evidence of either aortic or pulmonary incompetence.

In four of the cases the left auricle was described as being hypertrophied and dilated and in two as being hypertrophied. In eight, the right ventricle was mentioned as being hypertrophied, and in case 6, its wall measured 20 millimetres in thickness. In three, the tricuspid valves were thickened; the smallest measurement of the tricuspid orifice was 10·16 centimetres and the largest 15 centimetres. In two of the cases antemortem thrombi were found in the appendix of the left auricle; in one in the appendix of each auricle and in one in the appendix of the right auricle. The above changes all point to the enormous backward pressure which existed as a result of the marked stenosis of the mitral orifice in each case.

An analysis of all the post-mortem examinations on cases of advanced mitral stenosis would show almost identical changes, and one of the most extraordinary features of this disease is the comparative rareness of evidence of dilatation of the main trunk and orifice of the pulmonary artery.

The explanation, I repeat, must be found in the elastic recoil of the vessel when the tension is relieved by death or cardiac failure.

It must be evident that as mitral stenosis leads to hypertrophy of the right ventricle, the right ventricle must have much more work to do. If the right ventricle, then, has more work to do, and does it, there must be a corresponding increase in the tension of the pulmonary artery and its branches. That this is so, is shown by the changes I have already described in the branches of this vessel in the lungs. The effect appears at first to be produced on



the smaller branches, but later on, when the tension gets beyond a certain point, the main trunk and orifice yield to the excessive tension and pressure, and so the vessel becomes dilated and reflux of blood occurs into the right ventricle without there being any actual lesion of the pulmonary valves.

#### THE PHYSICAL SIGNS AND SYMPTOMS OF FUNCTIONAL PULMONARY INCOMPETENCE.

The most important physical sign of this condition is the presence of an early diastolic murmur. The position and character of this murmur will be best manifested by analysing the sixteen cases on which this paper is based, and by investigating the statements made by other observers.

##### Case 1 :—

(a). A whistling, early diastolic murmur was heard two inches to the inner side of the left nipple, and it was traceable upwards into the third left space. The bruit became fainter as it was traced upwards.

(b). An evenly sustained, diastolic, humming bruit of some harshness in quality, and commencing directly after the second sound and continuing nearly, if not quite, through the pause. The bruit occupies chiefly the right side area, and it is much louder immediately below the pulmonary valves (Dr. Goodhart) (*vide* fig. 1).

##### Case 2 :—

(a). A well marked blowing, early diastolic murmur was heard in the third and fourth left spaces, midway between the left border of the sternum and the left nipple line (*vide* fig. 2.) In the aortic area the second sound was loud and clear in character.

(b). In the pulmonary area, about three quarters of an inch outside the left border of the sternum, and over the third and fourth intercostal spaces, a distinct, soft, early blowing diastolic murmur following a rather loud second sound was heard (*vide* fig. 3). In the aortic area the second sound was sharp, and ringing in character.

## Case 3 :—

(a). A diastolic bruit was heard in the fourth left space, with reduplication of the second sound in the third left space.

(b). A faint, short diastolic bruit was heard in the third left space, coming immediately after a very accentuated second sound (*vide* fig. 4).

(c). In the second and third left intercostal spaces, just outside the left border of the sternum, an early diastolic murmur was heard (*vide* diagram 5).

Cases 4, 5, 6, 8 and 9. There was no clinical evidence of pulmonary incompetence.

Case 7. The second sound was very much accentuated in the second left space, where also a systolic and a faint diastolic murmur could be heard.

Case 10. In the second and third left intercostal spaces, midway between the left border of the sternum and the left nipple line a soft blowing, early diastolic murmur was heard (*vide* fig. 6). The aortic second sound was sharp, clear and accentuated.

Case 11. A faint, soft, blowing early diastolic murmur could be heard in the third left space, about two centimetres from the left border of the sternum, and 1.5 centimetres internal to the left nipple line (*vide* fig. 7). The aortic second sound was clear and distinct, and there was no visible pulsation of the carotid arteries.

Case 12. In the second and third left intercostal spaces midway between the left border of the sternum and the left nipple line, a soft early diastolic murmur was heard (*vide* fig. 8). No bruits were heard in the aortic area.

Case 13. A loud and accentuated second sound, followed by a soft, blowing, early diastolic murmur was heard in the second left space, just outside the left border of the sternum. The aortic sound was sharp and clear and no bruit could be heard accompanying or following it (*vide* fig. 9).

Case 14. In the third left space near the sternum the first sound was reduplicated, the second sound was loud and was followed by a faint, blowing, diastolic murmur (*vide* fig. 10).

## Case 15 :—

(a). A faint, soft, diastolic murmur was heard in the second left space, and could be traced downwards and outwards for about three centimetres (*vide* fig. 11).

(b). At the junction of the second left costal cartilage, and traceable downwards and outwards half-way towards the left nipple, a short, soft, diastolic murmur was heard (*vide* fig. 12).

Case 16. A diastolic murmur was heard in the second and third left intercostal spaces, just outside the left border of the sternum, and inside the left nipple line (*vide* fig. 13).

Pitt states that the most important signs of pulmonary incompetence are : Pulsation in the second and third left spaces close to the sternum, a condition which is partly the result of dilatation of the infundibulum and pulmonary artery, and a diastolic murmur audible on the left side of the sternum.

Balfour states that " In all cases of pulmonary diastolic murmur hitherto recorded there has always been, I believe, a loud systolic murmur preceding it." This is not my experience with the pulmonary diastolic murmur produced by functional incompetence of the valves resulting from dilatation of the pulmonary artery and orifice. Systolic basal bruits were only heard in two of the sixteen cases. It might be argued that if the pulmonary artery is dilated the condition necessary for the production of a fluid vein would be present and there should be a systolic bruit. But the probable reason of the absence of such a systolic murmur is that the pulmonary orifice is also dilated as well as the artery, so that the conditions for a fluid vein do not actually exist.

Sansom describes the bruit of pulmonary regurgitation as best heard over the second left intercostal space, and says it may be audible down the sternum to the apex of the left ventricle.

Broadbent describes the murmur of pulmonary regurgitation as being best heard in the third left intercostal space and that it may be conducted downwards.

Gibson describes the bruit of pulmonary incompetence as follows: " The murmur may be diffused over a wide area, but it appears invariably to have its maximum intensity in the second

left space. It is propagated towards the apex of the heart and its line of conduction is chiefly to the left of the sternum. The character of the murmur varies considerably. It may be soft and blowing or harsh and rasping." He records three cases of functional pulmonary incompetence.

In the first case (M.G., æt. 18), a short, sharp, high pitched, soft, diastolic bruit was heard immediately following the second part of the reduplicated second sound. It was heard over a small triangular area, two and a quarter inches by two inches along the lower border of the sternum from the lower border of the third costal cartilage to the upper border of the fifth costal cartilage. The murmur was quite different in character to the murmur of mitral stenosis, and there were no signs of aortic disease, so that it was looked upon as being a murmur indicating regurgitation from the pulmonary artery into the right ventricle "due to the increased pressure and consequent dilatation of the orifice, with relative and transient incompetence of the cusps."

In the second case (N. W., æt. 16) there was a diastolic murmur, soft in character, heard best in the third left intercostal space; the point of the maximum intensity was one and a half inches from the mid sternal line. The murmur followed a very distinct second sound. It could only be traced a short distance in each direction. There was no capillary pulsation; the pulse was not splashing in character. At the post-mortem examination the mitral valve was much stenosed. The pulmonary orifice was much larger than the aortic. The right ventricle was much enlarged. The aortic valves were competent.

In the third case (A. L., æt. 19), the first sound in the pulmonary area was pure, the second sound was much accentuated and was followed immediately by a short, soft, rather high-pitched diastolic murmur, quite different in character to the diastolic bruit audible in the mitral area. The point of the maximum intensity of the bruit was at the sternal end of the third left intercostal space, it could be traced a little upwards, but no bruit was heard in the carotids. In this case there was no evidence of aortic regurgitation.

*Fig. 100*

Barie classifies the causes of pulmonary incompetence thus :—

A. True pulmonary incompetence due to actual disease of the valves.

B. Functional or relative incompetence, in which its valves are not diseased, the incompetence being purely functional and due to dilatation of the pulmonary artery, this latter form being much rarer than the former.

Barie mentions that Pawruski and Gouget have recorded cases of functional incompetence of the pulmonary artery in some cases of mitral stenosis. He thus writes when describing the physical signs of this condition : “ Le signe capital est fourni par l'auscultation elle décèle la présence d'un souffle diastolique, le long du bord gauche du sternum dans le deuxième espace intercostal gauche.

Ce bruit “ semblable de tous points à celui qu'on rencontre dans les cas ordinaires d'insuffisance aortique.” (Stokes). “ Se propage dans la direction de l'artère pulmonaire c'est à dire le long du sternum jusqu'à vers le 4<sup>e</sup> espace intercostal. On la vu se propager encore jusqu'à vers la base de l'appendice xiphôide, à la façon de certaines insuffisances aortique ; dans un autre cas on le retrouvait même dans la région interscapulaire.”

Bramwell describes pulmonary incompetence as being attended by a diastolic murmur, heard in the pulmonary area best, traceable downwards and to the right, and often heard at the lower end of the sternum.

Gerhardt states that the diastolic bruit of pulmonary incompetence is appreciably augmented during expiration on account of the increased intra-ventricular tension.

From the analysis of the eleven cases in which a murmur was audible, it will be seen that the most characteristic physical sign of the condition, is, an early diastolic murmur immediately following the second sound. The second sound may be accentuated or reduplicated. The bruit is usually soft and blowing in character, it is often faint, it may be short, evenly sustained, or may continue nearly through the diastolic pause. It may be humming or whistling in character (*vide* case 1).

A glance at the diagrams will give the best idea of the distribution of the bruit, and one fact in particular becomes evident, viz., that the bruit is generally best heard not close to the left border of the sternum, as is usually stated, but midway between the left nipple line and the left border of the sternum. This is of great importance, and is probably one of the reasons why this complication of mitral stenosis is not more frequently recognised, as this particular region is not examined so systematically as it might be. If no bruit is heard in the second, third or fourth left intercostal spaces close to the sternum, the region just outside this area, which is midway between the left border of the sternum and the left nipple line, is frequently not examined at all, and so the bruit may be overlooked.

The distribution of the bruit was as follows: In the second and third left intercostal spaces five times, third and fourth left intercostal spaces three times, third left intercostal space three times, second left intercostal space once, and second, third and fourth intercostal spaces once. If the position of the bruit in each individual intercostal space is analysed the result is, third left intercostal space twelve times (out of thirteen), second space seven times, fourth space four times. From this it will be seen that the bruit is most frequently heard in the third left space, midway between the left sternal line and the left nipple line.

The pulmonary second sound was noted as being loud or accentuated in eight of the cases, and in two of the cases the second sound in the pulmonary area was reduplicated. The aortic second sound was described as being loud, clear or normal in six of the cases; in one it was faint.

The cardiac dulness was increased in thirteen of the cases, and this can be best seen by looking at the diagrams. The increase of dulness in most of the cases was upwards and to the right, indicating hypertrophy of the right ventricle.

The position of the cardiac impulse was dependent on the amount of hypertrophy and dilatation of the right ventricle. The character of the impulse did not present any peculiarity which would help in making a diagnosis of pulmonary incompetence.

Visible pulsation in the pulmonary area was only noted twice (cases 1 and 2). A diastolic thrill was felt in the third left intercostal space once (case 1). A bulging of the chest in the pre-cordial area was noted in four of the cases.

The pulse did not present any peculiar diagnostic character. The rate varied from 74 to 136, the average being 107. It was described as compressible 9 times, regular 6, irregular 4, full 3, soft 2, diastolic 2, weak 2, and intermittent 2. It was never described as being splashing or collapsing in character in any of these cases. Marked pulsation of the carotid and other arteries was absent.

<i>Albuminuria</i> was noted in	...	...	8 of the cases.
<i>Rales</i> were heard at the bases in	...	8	"
<i>Cyanosis</i> was noted in	...	7	"
<i>Edema of the Legs</i> was noted in	...	6	"
<i>Enlargement of the Liver</i> was noted in	...	5	"
<i>Epigastric Pulsation</i> was noted in	...	4	"
<i>Pleuritic Effusion</i> was noted in	...	4	"
<i>Ascites</i> was noted in	...	3	"
<i>A troublesome cough</i> was noted in	...	2	"
<i>Hæmoptysis</i> was noted in	...	2	"
<i>Hæmaturia</i> was noted in	...	1	"

From the above it will be seen that there are no symptoms or signs which are characteristic of functional pulmonary incompetence, with the exception of the early diastolic bruit which is audible most frequently in the third left intercostal space, and can be best heard at a point midway between the left border of the sternum and the left nipple line.

#### DIAGNOSIS.

A diagnosis of functional pulmonary incompetence can only be made after a most careful physical examination. There are no symptoms which are peculiarly diagnostic of this condition. Aortic regurgitation is the most likely lesion to be confused with it, and I shall therefore compare and contrast the symptoms of these two conditions.

A patient suffering from aortic regurgitation is usually pale, whereas a patient suffering from functional pulmonary incompetence is cyanosed. In eight out of eleven cases cyanosis, especially of the lips, was noticed.

*The pulse.*—In aortic regurgitation the pulse is usually splashing or collapsing in character. In pulmonary incompetence the pulse exhibits no special peculiarity, in fact, it is most likely to be characteristic of advanced mitral stenosis, as pulmonary incompetence does not usually occur unless the stenosis of the mitral valve is extreme and the condition is of long standing.

In aortic disease a characteristic feature is the marked visible pulsation of the carotid, temporal, brachial and other superficial arteries. No such marked or excessive pulsation is noticeable in pulmonary incompetence. In aortic regurgitation capillary pulsation can usually be demonstrated, but it is not present in cases of pulmonary regurgitation.

The position of the cardiac impulse does not help to distinguish these conditions. I have constantly noticed that when mitral stenosis and aortic regurgitation are associated together, the left ventricle does not hypertrophy to such an extent as it does when aortic regurgitation is the only lesion present, so that both in cases of aortic regurgitation and mitral stenosis, and of pulmonary incompetence and mitral stenosis, the position of the cardiac impulse will depend chiefly on the degree of hypertrophy of the right ventricle. If there is any difference it is slight, the impulse perhaps being a little lower and further to the left in the case of long continued aortic regurgitation and mitral stenosis on account of the slightly larger size of the left ventricle.

Palpation may reveal in the case of functional pulmonary incompetence a pulsation in the second and third left intercostal spaces, a condition which would not be likely to be associated with aortic regurgitation. Aortic regurgitation might be due to dilatation of the first part of the aorta, but in this case there would be pulsation in the second and third right intercostal spaces close to the right border of the sternum. It would, however, be most unusual to find aneurysm of the first part of the aorta associated with mitral



stenosis. A thrill is not common with regurgitation from either orifice.

The cardiac dulness in cases of pulmonary incompetence, secondary to mitral stenosis, is usually increased upwards and to the right, indicating considerable hypertrophy of the right ventricle, the increase of the dulness in these directions is much more likely to be associated with pulmonary than with aortic incompetence.

The fact of a diastolic bruit being heard on the left side of the sternum is put forward by some observers as an important indication of pulmonary rather than of aortic incompetence. The textbooks are very misleading on this point, for the early diastolic bruit indicative of aortic regurgitation is almost invariably described as being best heard in the third right intercostal space close to the sternum. I admit that this is so in a small proportion of the cases of aortic regurgitation, but I maintain that in nearly 90 per cent. of the cases the bruit is best heard in the third *left* intercostal space close to the left border of the sternum, and my experience is that the diastolic bruit of aortic regurgitation is not only best heard, but is first heard, in the third left space close to the left border of the sternum, and immediately beneath the lower border of the third left costal cartilage.

In pulmonary regurgitation an early diastolic bruit is heard best in the third left space, not close to the left border of the sternum, but midway between the left border of the sternum and the left nipple line. In all cases of advanced mitral stenosis this area should be most carefully auscultated for the early diastolic bruit of functional pulmonary incompetence.

The bruit of functional pulmonary incompetence is usually softer and less distinct than that of aortic regurgitation.

The aortic second sound in cases of pulmonary incompetence may be heard to be clear and distinct, whereas the pulmonary second sound is followed or replaced by the bruit.

In aortic regurgitation the bruit may be propagated into the carotid arteries, but in pulmonary incompetence no bruit is audible over these vessels.

Gerhardt has pointed out that the murmur of pulmonary incompetence is increased in intensity during expiration. This test was unfortunately not examined for in the cases I have reported.

Another observation which I cannot confirm or refute, is, that in pulmonary regurgitation an interrupted vesicular murmur may be heard during inspiration at the angle of the right scapula.

Broadbent writes: "Before venturing on a diagnosis of pulmonary regurgitation, it must be ascertained not only that the pulmonic sound is impaired, but also that the carotid throb and collapsing pulse are absent, and that the aortic second sound is unimpaired. No special train of symptoms can be attributed to pulmonary regurgitation."

Functional pulmonary incompetence must also be distinguished from pulmonary incompetence due to actual lesions of the pulmonary valves. Pulmonary incompetence from all causes is the rarest of all the valvular lesions of the heart. The pulmonary valves are hardly ever, if ever, affected by rheumatic endocarditis, so that, if, in a case of advanced mitral stenosis there is an early diastolic murmur indicative of pulmonary incompetence, it may be looked upon as being due to functional incompetence of the valves from dilatation of the pulmonary artery and its orifice. If, however, there is pyrexia and other physical signs and symptoms pointing to infective endocarditis, the probability will be that there are vegetations on the pulmonary sigmoid valves, rendering them incompetent. In connection with infective endocarditis affecting the pulmonary valves, I have observed, that in three out of the five cases which I have seen, there was undoubted evidence of gonorrhœa.

Some physicians are of opinion that this early diastolic bruit which is occasionally audible in the second, third and fourth left intercostal spaces is due to the mitral stenosis itself. I consider this interpretation is most unlikely and unsatisfactory, for I have never heard the presystolic or mid-diastolic bruits produced by mitral stenosis in these spaces and I fail to see why mitral stenosis should be considered capable of producing an early diastolic murmur in this position. Mitral stenosis without doubt is very occasionally indicated by an early diastolic murmur at the

point of the cardiac impulse, and between the impulse and the left border of the sternum, but there does not seem any justification for interpreting the early diastolic murmur which is audible most frequently in the third left space midway between the left border of the sternum and left nipple-line as being the direct result of the mitral lesion. That this murmur is the result of dilatation of the pulmonary artery and functional incompetence is a much more satisfactory explanation, and it is borne out by the very definite morbid changes which are found in the pulmonary artery and its branches in necropsies on cases of advanced mitral stenosis.

#### CONCLUSIONS.

1. That as a result of extreme mitral stenosis there is enormously increased tension produced in the pulmonary arteries.
2. That as a result of this increased pulmonary tension, the smaller branches of the pulmonary arteries in the lungs become dilated, hypertrophied and atheromatous.
3. That as a further result of this increased tension in the pulmonary artery, the main artery may become dilated and so may its orifice also.
4. That as a result of this dilatation of the pulmonary artery and its orifice, a reflux of blood may occur into the right ventricle through functional incompetence of the valves.
5. That the chief clinical indication of this functional pulmonary incompetence, is, the occurrence of an early diastolic murmur on the left side of the upper part of the chest, and, that the point where the bruit is best and most frequently heard, is, in the third left intercostal space midway between the left border of the sternum and the left nipple line.
6. That although there may be this clinical evidence of pulmonary incompetence, the post-mortem examination may fail to show any incompetence of the valves or dilatation of the pulmonary artery.
7. That the fact of there being no actual post-mortem evidence of regurgitation in some of the cases is no proof that the incompetence did not exist during life.

8. That the explanation of the last condition is as follows:— That the elasticity of the pulmonary artery has not been permanently injured by the increased pulmonary tension, and that as soon as the tension is relieved the vessel contracts to its normal size.

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## LIST OF CASES.

CASE 1.—C. S., æt. 36, music mistress, was admitted February 25th, 1891, under the care of Dr. Taylor (clerk, A. Leigh Allworth) for weakness and cough. There was a history of typhoid fever at sixteen years, and scarlet fever at twenty-one years. Since the typhoid fever she had always been short of breath, and in December, 1890, she had been compelled to give up her work.

*Condition on admission*, 25th February, 1891.—The cardiac impulse was in the fifth space, three-quarters of an inch internal to the nipple; the first sound was accentuated both at the apex and base, and the second sound reduplicated. There was a faint murmur with the first sound at the apex. Fine râles were heard on both sides of the chest, but the resonance was good. On March 2nd, 1891, the impulse was felt in the fourth and fifth spaces, and extended a little beyond the nipple-line. The cardiac dulness was not increased. A presystolic bruit was heard all along the fifth space, from about one and a half inches external to the nipple-line to the epigastric angle; it ended in a loud abrupt first sound. The second sound was accentuated in the second left space, and was also well heard at the apex, where it had a peculiar, short clicking character, rather difficult to describe. The first sound in the fourth space was short and sharp. The diagnosis was mitral stenosis, and she left the hospital on March 16th, 1891, much relieved.

(P.S. 116, 92. Clerk, L. Slater.)

She was readmitted under Dr. Pye-Smith on the 27th April, 1892. The cardiac dulness was limited above by the upper border of the fourth rib, and externally by the nipple-line. Internally it reached nearly to the mid-line of the sternum. The cardiac impulse was in the fourth and fifth spaces, and was felt as far out as the nipple-line. There was a very long, loud, rasping, presystolic bruit, audible best in the fourth space, three quarters of an inch internal to the nipple and there was also a thrill. Some râles were heard at the bases of the lungs. The urine contained a small quantity of albumen. *Mitral stenosis* was again diagnosed. A day or two before she went out Dr. Pye-Smith heard a faint diastolic murmur just to the left of the sternum below the third space. The heart-sounds were otherwise the same, and the thrill was still present. Presystolic pulsation in the vessels (? veins) of the neck was also noted. She left the hospital on May 23rd, 1892.

(G. 122, 1893, clerks, H. Hewetson, L. A. Parry, A. K. Matthews.)

She was readmitted under Dr. Goodhart on December 6th, 1892. The cardiac impulse was felt in the same situation as in April, and there was a diastolic thrill. Over the point of impulse a rumbling presystolic bruit was heard, running up to the first sound. *Two inches to the inner side of the left nipple, and traceable upwards into the third left space, a whistling early diastolic bruit was heard.* This bruit became fainter as it was traced upwards. There were troublesome fits of coughing, with occasional slight hæmoptysis, and moist râles were heard at the bases of the lungs. The urine contained albumen.

On December 9th, the following note was made in the report by Dr. Goodhart:—"She is rather blue about the lips and ears, suggesting mitral

stenosis. There is on palpation a grating first sound at the apex, followed by almost a click for the first sound, and then a faint diastolic thrill is perceptible; but as one passes up to the base, there is a much more pronounced systolic impulse over the third interspace to the left of the sternum, and after it a well-marked diastolic thrill. On auscultation at the normal impulse, there is characteristic presystolic bruit, short, sharp and grating, terminating in the first sound, the faint thrill being represented by a distant diastolic. But on passing upwards to the base, one comes finally upon a second loud, evenly-sustained diastolic humming bruit, of some harshness in quality, and commencing directly after the second sound, and continuing nearly, if not quite, through the pause. On carefully mapping out the area of the bruit, although it is not quite confined to it, yet it occupies chiefly the right side area, and it is much louder immediately below the pulmonary valves."

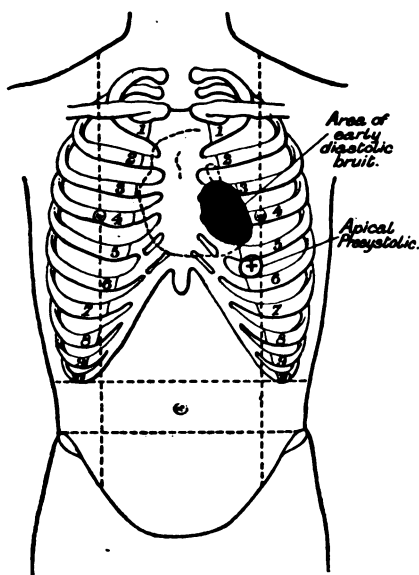


Fig. 1.

*A few days later, after ascertaining the observations made at previous admissions, Dr. Goodhart made another note that the previous observations seemed to point to the physical signs then present being produced by a dilated pulmonary artery.*

Signs of pleurisy were found soon afterwards on the left side, and on January 23rd 426 cubic centimetres of clear serous fluid were drawn off from the left side of the chest. The fluid was *acid*, specific gravity 1011, and contained twenty-four parts per 1000 of albumen.

The patient improved considerably after this, and left the hospital on February 23rd, 1893, the bruits remaining the same.

She was readmitted on March 8th, 1893, for bronchitis and vomiting.

*Condition on admission.*—Pulse 90, respiration 30, temperature 99°, a diastolic thrill was felt over the cardiac impulse in the fifth space, and also over the pulmonary area. On auscultation at the apex, there was a loud bruit, commencing almost at the beginning of diastole, and running quite up to the first sound. A very loud and high-pitched diastolic murmur was heard in the pulmonary area. Râles and rhonchi were heard on both sides of the chest. There was no œdema of the face or feet. The urine was loaded with urates, specific gravity 1030, and contained five parts per 1000 of albumen. She died suddenly on March 13th, 1893.

*Post-mortem*, 102, 1893, by J. H. Bryant.—The body was wasted, and the face had a haggard expression. The brain was not examined. The thyroid gland was healthy, but there was some extravasation of blood around the lower part of the neck at the level of the lower border of the thyroid. The pleura over the lower lobe of the left lung was thickened, opaque, and greyish-white in colour. 340 cubic centimetres of fluid were found in the right pleural cavity, and 280 cubic centimetres in the left. Both lungs were red, tough, and firm from chronic congestion (red induration). The lower lobe of the left lung was compressed, of a dull, slate-grey colour, and readily sank in water. *The branches of the pulmonary arteries in the lungs were atheromatous, more especially on the right side and in the right lower lobe.* The larynx, trachea, bronchi, and bronchial glands were healthy. The pericardial sac contained about 30 cubic centimetres of serous fluid. The heart was not weighed (it being left with the lungs to form a museum specimen). The right ventricle was empty, and there was a little post-mortem clot in the right auricle. The right side of the heart was enlarged, and the wall of the right ventricle much thickened, measuring 13 millimetres. The muscle was of good colour, and the tricuspid valves were healthy. *The pulmonary valves appeared to be very thin and loose, and each corpus arantii was considerably thickened. The free edges of these valves were slightly curled towards the ventricle.* The walls of the left auricle were a little hypertrophied, and the endocardium was thickened. The mitral orifice was very small—only just admitting the tip of the little finger. The mitral valve was funnel shaped, the opening being at the apex. The valve was much thickened, the chordæ tendinæ were shortened and thickened, and some of the muscoli papillares were directly continuous with the edge. It measured 23·8 millimetres. The aortic valves were healthy. On opening the pericardial sac, *the pulmonary artery appeared to be dilated, and it felt much thicker than normal—almost as thick as the aorta (Drs. Goodhart and Shaw confirmed this).* *The pulmonary orifice measured 91·5 millimetres, and 20 millimetres above the orifice; the circumference of the vessel was 101·6 millimetres.* On tracing up the branches in the lungs, a number of patches of atheroma were seen, especially on the right side and in the vessels of the middle and lower lobes. There was some atheroma of the thoracic and abdominal aorta. The liver weighed 994 grammes, and was soft; it showed no sign of backward pressure. The spleen was small and hard, it weighed 76·2 grammes. The kidneys weighed 203 grammes. The capsules stripped off easily, but the surfaces were scarred and irregular. The uterus was large, and the cervical canal large and patent. There was a cyst the size of a pigeon's egg in the left ovary. The right ovary was bound down by old adhesions to the broad ligament. The remaining viscera were healthy.

CASE 2.—F. N., æt. 40, under the care of Dr. Taylor (clerk, L. C. Martin) was admitted on December 27th, 1897, for dropsy and dyspnoea. Her father and mother were alive and healthy. One sister died of dropsy. When fifteen years of age she had an attack of rheumatic fever. She did not remember any other serious illness, but she stated that she had attacks of palpitation when ten years old. In September, 1896, she first noticed swelling of her thighs, followed by swelling of her legs and ankles. Rest in bed relieved her, and she remained fairly well until June, 1897, when she was admitted into Mary ward under Dr. Pye-Smith on June 30th.

*Condition, July, 1897 (P. S. 223).—Circulatory system:* pulse 88, regular, soft and compressible. The cardiac impulse was visible in the fifth left space just internal to the nipple-line; it was diffuse in character and could be seen two inches internal to the nipple-line. No thrill was felt. Dulness, *vide* diagram. At the impulse a short rumbling presystolic murmur running up to a loud first sound, which was followed by a faint systolic murmur, was heard. In the third and fourth left spaces (*vide* diagram) a well-marked

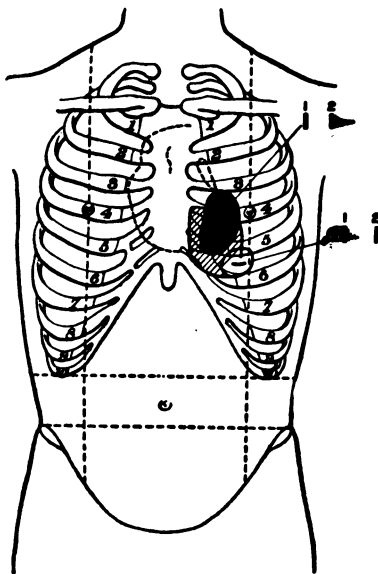


Fig. 2.

*blowing early diastolic murmur was heard.* In the aortic area the second sound was loud and clear in character. There was no visible pulsation of the carotids or other arteries.

*Condition on admission, December, 1898.*—Temperature 98°, pulse 110, respiration 30. Anæmic. She was suffering great pain and was unable to pass her urine. Her legs were very swollen and œdematous. *Circulatory system.*—Pulse 110, regular, soft and compressible. The cardiac impulse was diffuse in character and best visible in the fifth and fourth spaces just internal to the left nipple line. Pulsation was also visible in the third left space above and



in the second left space close to the sternum. A retracting impulse in the epigastrium synchronous with the carotid beat was also noted. The above mentioned pulsations were confirmed by palpation. No thrill was felt. The cardiac dulness was limited above by the lower border of the third rib, internally by the left border of the sternum, below by the fifth rib, and externally by a line drawn from the left nipple to the impulse. At the apex a loud systolic murmur was heard, traceable outwards into the left axilla, and nearer the sternum a rumbling diastolic murmur running close to the first sound but not quite up to it. *In the pulmonary area about three-quarters of an inch outside the left border of the sternum and over the third and fourth spaces (vide diagram) a distinct, soft, early blowing diastolic murmur,*

of these vessels. The pericardium was healthy. The heart weighed 397 grammes. There was a thin layer of fat over the right ventricle. The coronary arteries were thickened and atheromatous. The right ventricle was full of post-mortem clot. A large ante-mortem thrombus was found in the appendix of the right auricle. The right ventricle and auricle were dilated, and the wall of the right ventricle was hypertrophied. The tricuspid orifice measured 10·16 centimetres. The valve was not thickened. *The pulmonary artery appeared to be much larger than normal, for it seemed almost double the size of the aorta. The pulmonary orifice measured 8 centimetres, and 12 millimetres above the orifice the circumference of the lumen was 10·16 centimetres.* The pulmonary valves were not thickened, and appeared normal. A large ante-mortem thrombus was also found in the appendix of the left auricle. The left ventricle and auricle were hypertrophied and dilated. The endocardium of the left auricle was much thickened. The mitral valve was very thickened and calcareous and the orifice would only just admit the tip of an index finger; it measured 3·81 centimetres in circumference; it was buttonhole in shape. The chordæ tendinæ were thickened and shortened; some calcareous process from the valve extended into the muscle of the left ventricle. *The aortic valves were normal.* The stomach, œsophagus, and intestines were congested. 1847 cubic centimetres of clear yellow serous fluid was found in the peritoneal cavity. The liver weighed 1474 grammes. The surface was slightly irregular and showed numerous patches of perihepatitis, and on section it presented an advanced nutmeg condition. *Pancreas* very hard. *Spleen*, 226 grammes, very hard. *Kidneys*, 340 grammes, scarred and showing recent yellow infarcts.

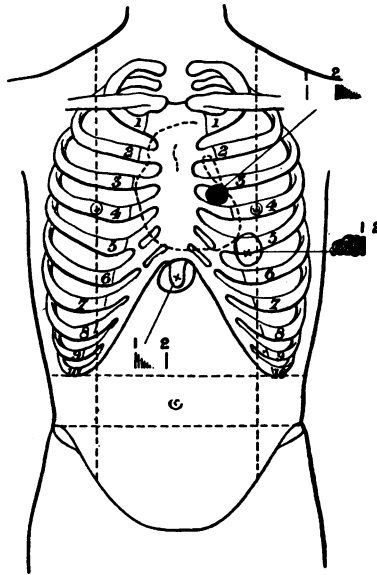
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CASE 3.—E. S., æt. 41 years, sack sewer, was admitted 15th May, 1895, under the care of Dr. Goodhart (clerk, R. B. Stamford), for shortness of breath and swelling of legs. She was a widow, and had never had any children, but had had one miscarriage. She had always worked very hard. She had been in the hospital seven times before, chiefly for cardiac trouble. Her first admission was under Dr. Pavy, in 1883, for intestinal obstruction, and while under treatment she developed her first attack of rheumatism.

She was readmitted under Dr. Goodhart, February 8th, 1888, and remained in the hospital until March 20th, 1888. She then had a presystolic bruit and thrill at the apex, and she had rheumatic pains while in the hospital. The diagnosis made was mitral stenosis and bronchitis. (G. 117, 1888. Clerk, J. V. Blackford.)

She was readmitted January 11th, 1889, under Dr. Taylor, and remained in until January 30th, 1889. There was the same presystolic bruit and thrill, and in addition, she had a reduplicated second sound in the pulmonary area. Mitral stenosis and pleurisy were diagnosed. (C. 171, 1889. Clinical, J. M. Gill.)

Her next admission was May 30th, 1893, under Dr. Pye-Smith, when she remained in the hospital until July 2nd, 1893. Mitral stenosis, tricuspid regurgitation, tricuspid stenosis and enlarged liver were diagnosed. On June 12th, 1893, *Dr. Pye-Smith heard a diastolic bruit in the fourth left space with reduplication of the second sound in the third left space.* On June 23rd, 1893, there was a systolic bruit at the ensiform cartilage, a presystolic bruit at the apex, and a faint short diastolic bruit in the third left space coming



*immediately after a very accentuated second sound.* (P.S. 103, 1893. Clerk, R. Henderson.)

She was readmitted on July 16th, 1894, and remained in until July 25th, 1894, under Dr. Shaw. The bruits were then presystolic (no thrill) and systolic at the apex, systolic at the ensiform cartilage, while there was a reduplicated second sound heard to the right of the sternum, and an accentuated second sound in the pulmonary area. The liver reached 5 centimetres below the costal margin. The diagnosis was acute supervening on chronic bronchitis, mitral stenosis and tricuspid regurgitation. (C. 336, 1894. Clinical, W. E. Robinson.)

She was admitted again, October 27th, 1894, under Dr. Taylor, with signs of extreme backward pressure, and venesection was performed, which greatly relieved her. She had a long presystolic bruit at the apex, with no recognisable second sound, and there was a squeaking systolic bruit over the ensiform cartilage. Her fingers were clubbed, the liver was three fingers' breadth below the ribs, and her feet and ankles were œdematous. The diagnosis made was mitral stenosis, tricuspid regurgitation and acute bronchitis. She went out again February 20th, 1895. (C. 1, 1895. Clinical, A. Salter.)

*Condition on admission, 15th May, 1895.* Temperature 99°, pulse 120, respiration 56.—There was some œdema of the legs and ankles, and some ascites. Pulse regular, low tension; no pulsation of the veins of the neck. The cardiac impulse was very diffuse, and was felt in the fifth and sixth spaces, extending in the sixth space as far as five centimetres outside the nipple-line. There was no thrill. Some epigastric pulsation was noticed. The cardiac dullness extended above to the fourth rib, externally to the nipple-line, and internally

to the mid-sternal line. At the impulse there was a loud, rough presystolic murmur running up to an accentuated first sound, followed by a soft systolic murmur, with no recognizable second sound. Traced out into the axilla, the first sound was replaced by a systolic bruit. Over the lower end of the sternum and in the epigastrium, a softer blowing systolic murmur was heard, followed by a second sound. The murmur could be traced to within 4 centimetres of the right nipple. In the pulmonary and aortic areas an early diastolic murmur was heard, not distinguishable to the right of the sternum (?). Numerous râles and rhonchi were heard all over the chest. The liver extended 5 centimetres below the costal margin, and the urine contained a trace of albumen. On May 17th, the early diastolic murmur in the pulmonary area had disappeared; but it was just audible again on May 18th. On May 18th it was again not heard. On May 23rd Dr. Goodhart heard a systolic bruit in the pulmonary area. On May 29th the early diastolic murmur in the third left space near the sternum was again heard (J. H. B.). It was noted again on June 5th, but was not mentioned after. Patient left the hospital on July 17th, 1895.

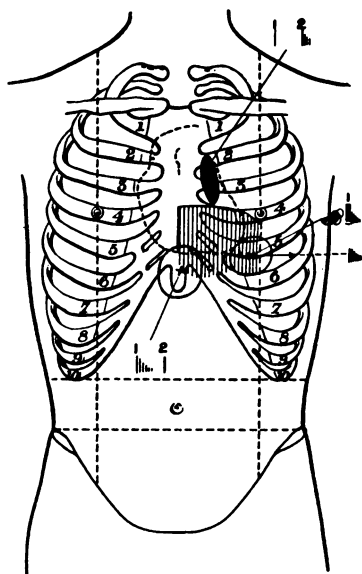


Fig. 5.

She was readmitted September 20th, 1895, under Dr. Pitt (C. 493, 1895. Clinicals, A. V. Clarke and E. Erskine Henderson).

*Condition on admission.*—Pulse 100, irregular and very feeble. Much cyanosis. Veins of neck pulsating, considerable œdema of the legs and feet. Cardiac dulness was the same as on May 15th, 1895. The impulse was diffuse in the fifth space one inch internal to the left nipple. No thrill. Systolic and presystolic murmurs at the apex, no second sound. Systolic murmur audible at the base. There were râles and slightly impaired resonance at both bases. The

liver was soft, pulsating and tender, and extended 5 centimetres below the costal margin. The urine contained a trace of albumen. On October 2nd the bruits were a presystolic at the apex where no second sound was audible, and a systolic bruit over the lower end of the sternum where the second sound was heard. A systolic bruit was heard at the base and thought to be the tricuspid bruit transmitted. On November 12th she had another attack of bronchitis and died on November 18th, 1895.

*Autopsy* 459, 1895, by J. H. Bryant.—The body was fairly well nourished and the face was cyanosed. The pleura covering the right lower lobe of the lung was very much thickened, and there was evidence of old pleurisy over the left lower lobe and also over the posterior surface of the upper lobe. The bronchial glands were enlarged and the lungs were tough and of a reddish-brown colour. There was very little œdema and no infarcts. The trachea and bronchi were congested. There was no pericarditis. The heart weighed 350 grammes and the right side was hypertrophied and a little dilated. The wall of the right ventricle measured 9·5 millimetres in thickness and the muscle was good. The right auricle was a little dilated. The tricuspid valve measured 10·4 centimetres and the pulmonary orifice 7·62 centimetres. The tricuspid valve was slightly thickened. The left ventricle was not hypertrophied, but the left auricle was both hypertrophied and dilated. There were a few recent vegetations on the auricular edge of the mitral valve, which was 3·17 centimetres in circumference and was much thickened and calcareous. The chordæ tendinæ were short, thickened and adhering to each other. The aortic valves were healthy. *The pulmonary artery was thickened and dilated; it was almost as thick as the aorta. 2·5 centimetres above the pulmonary valves it measured 8·25 centimetres in circumference when laid open. The two main branches were thickened and atheromatous, and the small branches in the lungs were thickened, atheromatous and dilated, and stood out prominently from the cut surface of the lung, with open lumina.* The stomach was congested, and there were a few submucous petechiæ. The duodenum was congested. There was no ascites. The liver was soft and weighed 1304 grammes; it was not typically nutmeg. The spleen was hard and weighed 170 grammes. The kidneys weighed 340 grammes; they were very hard and congested and the cortex was scarred in places and the capsule thickened.

CASE 4.—Eliza W., æt. 32, admitted June 9th, 1894, under the care of Dr. Pitt (clerk, C. L. Hopkins), for anasarca and dyspnoea. Her father had twice had rheumatism, and one of her brothers had also suffered from rheumatism. In 1889, she had an attack of rheumatism, and another in July, 1893. At this time the cardiac impulse was found to be just inside the left nipple-line in the fifth space. The cardiac dulness was normal. There was a harsh presystolic bruit at the apex, and the aortic and pulmonary sounds were accentuated. A diagnosis of acute rheumatism with mitral stenosis was made. She had been married fourteen years, and had had five children. On December 11th, 1893, she was admitted for marked dropsy. In addition to the mitral bruit, a tricuspid regurgitant murmur was heard. There was no bruit at the base, but the second sound was reduplicated. She went out relieved at the end of the month.

When admitted on June 9th, 1894, she was in a collapsed condition, and was very dyspnoic and cyanosed. The pulse could not be felt at the wrist.

There was very extensive œdema of the legs, and there was ascites. The heart was beating 132 to the minute. On the following day the apex beat was found to be diffuse and irregular, and was in the sixth space. Four centimetres outside the nipple-line there was marked epigastric pulsation. The cardiac dullness commenced above at the third rib, to the left it was limited by a line 13 millimetres to the right of the right border of the sternum, and outwards by a line 25·4 millimetres to the left of the left nipple-line. There was a presystolic thrill at the apex. A rough localised presystolic bruit was heard at the cardiac impulse, followed by a blowing systolic murmur, which could be traced outwards. In the aortic area the sounds were faint. The urine was 1020, and contained a trace of albumen. There was marked ascites, and in consequence the liver could not be felt. On the 11th she was better. She was so dyspnoëic, however, that she could not lie down. On the 13th she was much about the same in the morning; but about 7 p.m. she was very much worse, becoming gradually cyanosed and breathless. The hands and forehead became cold. The heart beat was rapid, fluttering, irregular and uncountable. The bases of the lungs were resonant. Dr. Pitt suggested that the sudden change was due to pulmonary thrombosis. At 5 a.m. she became worse; an injection of Liq. Strychnine  $\text{m.v.}$  was given, and oxygen was administered. Towards evening she became better. She again became worse on the 14th, and died at 12.15 p.m.

*Post-mortem*, 234, 1894.—The *post-mortem* was made by Dr. Perry fourteen hours after her death. There was much œdema of the lower extremities. The right pleural cavity contained 426 cubic centimetres, and the left 341 cubic centimetres of serous fluid. There were filamentous adhesions on the right side. The lungs were in a condition of brown induration, and there were definite hæmorrhages into the substance at the lower part of both lower lobes. The heart weighed 482 grammes. Both sides were large and dilated. *On the right side the pulmonary valves measured 11·4 centimetres in circumference, and therefore there was doubtless pulmonary regurgitation. In the artery were atheromatous patches, and in the left branch of it a small piece of ante-mortem clot, which, however, did not at all occlude the lumen of the vessel to any important extent.* The tricuspid orifice was 15 centimetres in circumference. On the left side the mitral orifice just admitted the index finger, and the aortic valves, which were competent to all appearances, measured 6·35 centimetres in circumference. The aorta was therefore small. There was ante-mortem thrombus in both the right and left appendices auriculæ. There was also an ante-mortem thrombus in the abdominal aorta, just above its division into the common iliaes. The left ventricle was very slightly enlarged. The œsophagus, stomach and intestines were a good deal congested. The liver weighed 1673 grammes, and was nutmegged. The spleen weighed 170 grammes; it was hard and scarred, and showed an organising infarct. The kidneys weighed 397 grammes; they were hard and scarred from infarction.

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CASE 5.—Fred. J., æt. 20, photographer, was admitted under the care of Dr. Washbourn, on December 8th, 1897, for shortness of breath. Five years ago he had some pain and swelling in his joints. He first noticed swelling of his hands and arms at the end of the bicycle racing season; about fourteen months before, this was followed by shortness of breath, and he had to give up bicycling.

*Condition on admission.*—Pulse very rapid and irregular. The cardiac impulse was very diffused and extended from the nipple line in the fifth space to the border of the sternum. There was no thrill. The cardiac dulness commenced above at the third rib, extending outwards to the left nipple line and inwards to the mid sternal line. There was a systolic bruit heard at the apex which could be traced inwards to the sternum and outwards to the anterior axillary line. The sounds at the base appeared to be normal. On December 11th, the pulmonary second sound was noted as being accentuated. On the 15th, he complained of tonsillitis. On the 16th, a pleuritic rub was heard at the left base behind. On the 21st, mitral and tricuspid regurgitant bruits were audible. On January 4th, pleuritic effusion was noted. On January 7th, purpuric spots were noticed on the thighs and abdomen. The urine was 1022 and contained blood and albumen. On January 10th, an apical diastolic bruit was heard. He was much worse; the purpuric rash had spread, and he died at 6.25 a.m. on the 11th.

Pulmonary regurgitation was not diagnosed, nor in fact was mitral stenosis. He was thought to be suffering from mitral and tricuspid regurgitation.

*Post-mortem* 19, 1898.—The post-mortem examination was made seven hours after death. Rigor mortis was well marked. There was considerable œdema of the legs. Numerous petechiæ and ecchymoses were seen over the abdomen, back, and upper part of the thighs. The thyroid was uniformly enlarged. Each pleural cavity contained about a litre of clear, pale serous fluid. There was recent pleurisy on the edge of the left lung. The pleuræ were thickened and adherent over the base and posterior surface of the left lower lobe. Both lower lobes were a good deal compressed. Both lungs were tough and in a state of red induration. *The branches of the pulmonary arteries in the lungs were thickened and atheromatous, standing out prominently with open lumina from the cut surface of the lung.* There was no pericarditis. The heart weighed 596 grammes. There was a slight amount of fat on the anterior surface of the right ventricle. The coronary arteries were dilated. The right ventricle measured 11.43 centimetres across at the base, its wall was hypertrophied and the cavity was dilated. The tricuspid orifice measured 10.16 centimetres, and the valve was a little thickened. The right auricle was dilated; the pulmonary veins were normal. *The pulmonary artery was dilated.* There was some ante-mortem blood-clot in the appendix of the left auricle. The left auricle was hypertrophied and dilated. The endocardium was thickened. The left ventricle was dilated. The mitral valve was thickened and calcareous and its orifice was much narrowed and measured five centimetres in circumference. The chordæ tendinæ were thickened and shortened. The aortic valves were slightly thickened and adherent, but appeared to be competent. The stomach was contracted; its mucous membrane was congested. There were several cubic centimetres of clear serous fluid in the peritoneal cavity. The liver weighed 1843 grammes; on section it presented a typical nutmeg appearance. The spleen weighed 368.5 grammes; it was very hard; the suprarenal capsules were normal. The kidneys weighed 411 grammes; the capsules were a little thickened; both kidneys were extremely hard and congested. There was no sign of embolism; there was no macroscopic appearance of nephritis. The hæmaturia was probably due to congestion.

CASE 6.—Henry W., æt. 22, a hawker, was admitted on January 19th, 1894, under the care of Dr. Taylor (clinical clerk, R. G. Dellbruck) for palpitation of the heart and pain in the chest. He had previously been in the hospital on two occasions, first under Dr. Hale White and then under Dr. Washbourn. A diagnosis of mitral stenosis and regurgitation was made on both occasions. His father died of heart disease and his mother suffered from palpitation. When eight years of age he suffered from scarlet fever. No history of rheumatism or chorea could be obtained. In the summer of 1892 he began to have attacks of cardiac syncope. In the winters of 1892 and 1893 he had bronchitis. For the six months previous to his admission he had had dropsy.

*Condition on admission.*—Pulse 92, full and regular. The left side of the chest near the sternum was a little more prominent than the corresponding part on the right side. The impulse was diffuse and was in the fifth left space, just to the left of the left nipple line. The cardiac dulness commenced above at the fourth rib, extended inwards to the right border of the sternum and outwards to a line 2·5 centimetres external to the left nipple line. In the sixth space, about 4 centimetres outside the impulse a loud, rumbling presystolic murmur running up to a loud first sound, which was immediately followed by a systolic murmur, was heard. At the point of the cardiac impulse a loud systolic murmur was heard. The second sounds at the base were accentuated. There were no basal bruits. There was no marked pulsation of the carotids or other arteries. On January 27th, he had two sharp attacks of palpitation. On February 9th, on auscultating just outside the nipple in the fifth left space the sounds had a different character on light and firm pressure of the stethoscope. On light pressure a booming sound was heard which appeared to occupy the whole rhythm of the heart and was heard alone during diastole, a blowing murmur being heard during systole. On firm pressure a triple sound was heard, the first sound appearing to be reduplicated, though the heart was beating too rapidly to be sure that it was the first sound that was reduplicated. The booming sound disappeared, but the blowing systolic bruit retained its character. On February 23rd Dr. Taylor writes:—"Systolic murmurs as above described, marked systolic thrill over impulse, and nearly to sternum. Diastolic murmurs as before in impulse and in axilla." Pulsating liver. He lies drowsy, pallid, dusky, with rapid breathing—36. Pulse small, regular, feeble; not specially indicative of any valvular disease. On the 24th he became slightly jaundiced, and was much worse. He died at six p.m.

*Post-mortem* (by J. H. Bryant).—Performed twenty hours after death. Rigor mortis was slight. There was marked hypostasis of the back. There was no recent pleurisy, but there were old fibrous filamentous adhesions over the posterior surfaces of both lungs. The lungs were tough and hard, in a condition of marked red splenization. *On section, the pulmonary arteries stood out very prominently from the cut surface; they were dilated, very much thicker than normal, showing marked atheromatous changes, appearing in fact almost as thick as similar-sized bronchial tubes.* There was no pericarditis. The heart weighed 510 grammes. On opening the pericardial sac and looking at the anterior surface of the heart it appeared to be almost entirely made up of the right ventricle, the left ventricle only just being visible, the right ventricle at least consisted of five-sixths, the left only one-



sixth of the two ventricles together. The wall of the right ventricle measured two centimetres in thickness. The right auricle was also hypertrophied and very much dilated. *The pulmonary orifice measured seven centimetres, just above the orifice the artery measured 8.8 centimetres and had the appearance of being considerably dilated, and was much larger than the aorta.* The tricuspid orifice measured 14 centimetres. The left ventricle was very small and its wall measured just under 13 millimetres in thickness. The curtains of the mitral valve were very much thickened and extremely hard. The tendinous cords were short and very thick. The mitral orifice would only admit the top of the little finger and measured 37 millimetres in circumference. The endocardium of the left auricle was thickened. The aortic valves were healthy. As above mentioned, the pulmonary artery was dilated just above the valves, and atheroma was marked in the branches commencing in the two first divisions.

CASE 7.—Julia D., æt. 27, was admitted on October 23rd, 1890, under the care of Dr. Hale White (clinical clerk, A. H. Trevor), for her third attack of acute rheumatism. Seven years before she was under the care of Dr. Taylor for mitral stenosis, and nine years before this she was also in the hospital for acute rheumatism and pericarditis. On this occasion a systolic apical murmur was also noted. Pain and swelling of her right elbow appeared on October 17th, and kept her from her work; on the two following days other joints were affected, and she kept her bed until the 23rd, when she came to the hospital, and was admitted.

*Condition on admission.*—Pale and emaciated, lips livid; the pulse was weak, rapid and compressible. The cardiac impulse was seen and felt in the fifth left intercostal space in the nipple line. No thrill could be felt. The cardiac dulness was increased to the right border of the sternum, above to the third rib, and to the left to the left nipple line. On auscultation, at the point of the cardiac impulse a loud presystolic murmur was heard running up to and continuous with a loud first sound. At the base, a blowing systolic murmur was heard, chiefly on the right side of the sternum. The second sound was accentuated in the second left intercostal space. Rhonchi and râles were heard at both bases. The tongue was covered with a thick brown fur. The liver was felt below the right costal margin. The urine was 1026; it did not contain albumen or sugar. On October 29th, Dr. Hale White heard a pleuritic rub in the left axillary region. On November 5th, a systolic bruit was heard at the apex, which was traceable outwards into the axilla. On November 7th, the following report was made:—"Patient's pulse was very slow yesterday afternoon (50) (? due to digitalis). This morning it is 56, and on auscultation it corresponds to the cardiac beats. A systolic murmur, and also a presystolic murmur can be heard at the apex, but 25 millimetres outside the apex nothing can be heard but a very sharp loud first sound, and occasionally a very feeble second sound. The systolic murmur can be heard all over the base, but is clearest at the left edge of the lower part of the sternum. The second sound is very much accentuated in the second left space, where also a systolic and a faint diastolic murmur can be heard. The first sound can be plainly heard by listening at the patient's back through the flannel vest." November 9th.—A mid-diastolic bruit was heard at the apex, and a diastolic thrill was felt. November 10th.—The patient became very dyspnoic, and died at 10.15 p.m.

The *post-mortem* was performed by Dr. E. W. Goodall, twenty-nine hours after her death. The body was pale and thin. There was slight œdema of the legs. The pleura was thickened over the right upper lobe. There was recent pleurisy involving the rest of the right lung and the whole of the left. The lungs were in a condition of brown induration. There were a few recent infarcts, the largest being in the left lower lobe behind. The pericardium was universally adherent, but could be stripped off in front. The heart weighed 510 grammes. There was some ante-mortem clot in the appendix of the right auricle, and amongst the papillary muscles of the right ventricle. The right ventricle and auricle were both dilated and hypertrophied. The tricuspid valve was much shortened and thickened, the circumference of its orifice was 12·7 centimetres. *The pulmonary orifice measured 7·6 centimetres. The pulmonary artery was dilated, and was much larger than the aorta. The intima was thickened, and the larger branches showed marked atheromatous changes.* The right ventricle showed several fibroid patches in the septum ventriculorum and at the apex. The left auricle was considerably dilated and hypertrophied, and the endocardium was much thickened. The left ventricle was slightly dilated and hypertrophied. The mitral orifice was much narrowed and the valve button-hole. The orifice would only admit the tip of the little finger. The valve segments were rigid, very short and thick. The chordæ tendinæ were glued together and short, and there was marked fibroid change in the upper parts of the papillary muscles. The aortic orifice measured 5·6 centimetres. The valves were quite competent, although the valve segments themselves were certainly thickened and also the corpora-arantii. Liver, 1474 grammes; marked nutmeg appearance on section. Spleen, 142 grammes, softer than usual in a mitral case. The capsule was thickened. Kidneys, 340 grammes; very firm; the remains of a few old infarcts were seen here and there in each organ.

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CASE 8.—Eliza S., æt. 25, admitted on July 1st, 1888, under the care of Dr. Perry (clinical clerk P. Paget) for dyspnœa. She was first admitted on January 13th, 1888, for mitral stenosis, tricuspid regurgitation, bronchitis and pregnancy, and went out on February 22nd relieved. She was again admitted on March 7th, and was discharged relieved on April 13th. There was a history of rheumatic fever in 1882.

*Condition on admission.*—She was pale and dyspnoic, but was not cyanosed. The pulse was 90, irregular, intermittent and compressible. The cardiac impulse was diffuse and could be seen in the fifth, sixth, and seventh spaces, in and outside the left nipple line. A well marked thrill could be felt at the apex. The cardiac dulness was increased a little in all directions. On auscultation, a murmur of maximum intensity at the apex was heard, and it could be traced outwards into the axilla: it was of a sharp blowing character and occurred with the first sound. There was no presystolic bruit. There was marked œdema of the legs and back and many purpuric spots were seen on the legs. The urine was 1020; it did not contain any albumen. She was put on ten minim doses of Tinct. Digitalis. On the 22nd, the pulse was better. It was noticed that she was slightly cyanosed. On the 23rd, she was more cyanosed and was very orthopnoic, and a well marked presystolic bruit was heard. The chest was explored for fluid, but none was found. On the 24th, as she was restless, an injection of Morphia gr.  $\frac{1}{4}$  was given, and after this

she vomited and her pupils became contracted; at 2.15 p.m. she was very cold and almost pulseless; she died at 2.15 p.m.

*Post-mortem*, 255, 1898.—The post-mortem was performed fifteen hours after death. The body was much cyanosed, there was marked cedema of the legs, and there were a few purpuric spots on the legs. The pleura was thickened and adherent over the posterior surface of the right lung. The lungs were in a condition of brown induration. There was a small infarct in the lower and outer part of the left upper lobe. The pericardial sac contained 313 cubic centimetres of brownish serous fluid. The pericardium covering the heart was thickened. The heart weighed 397 grammes, and it was broadened by the increase in the size of the right ventricle. The left auricle was much hypertrophied and dilated and its walls were tough and leathery. The mitral valve was converted into a calcareous ring, just admitting the end of the index finger; there were a few vegetations on the ring. The left ventricle was slightly hypertrophied; it was not dilated. The edges of the aortic valves were thickened and rolled. It seemed as though there must have been some regurgitation, but no murmur was ever heard, and the left ventricle was not much enlarged. The right auricle and ventricle were dilated and hypertrophied. The tricuspid valve had thickened edges and allowed regurgitation. It measured 10 centimetres across. *The pulmonary artery was much larger than the aorta, was much thicker, and had atheromatous patches in it. It measured 7.6 centimetres across against 5.7 centimetres of the aorta. The liver weighed 1758 grammes, it was nutmegged and fatty in appearance. The spleen weighed 255 grammes, it was hard. The kidneys weighed 283 grammes. There was some scarring from infarcts.*

CASE 9.—David L., was admitted on March 2nd, 1887, under the care of Dr. Taylor (clinical clerk R. G. Hicks) for dyspnoea. He had twice suffered from rheumatic fever; first of all in 1848 and again in 1871. About Christmas he first noticed a little difficulty in breathing and since that time it gradually became worse. About a fortnight before his admission to the hospital he experienced one night a good deal of difficulty in getting off his stockings, and found that his legs and ankles were much swollen.

*Condition on admission.*—He was a fairly healthy-looking man; his eyelids were a little puffed and there was marked cedema of the legs and feet. The pulse was 98, full, soft and regular. The cardiac impulse was in the sixth space 2.5 centimetres outside the nipple line. The impulse was rather forcible. There was distinct systolic retraction in the epigastrium. A loud systolic bruit was heard at the apex and it could be traced outwards into the left axilla, but it could not be heard behind at the angle of the scapula. The respirations were 28; râles and rhonchi were heard at both bases. The liver was enlarged and tender and its edge could be felt at the level of the umbilicus. The urine was 1017, and it contained albumen. On March 3rd, there was no albumen in the urine, and the cedema was less. On March 5th, he had a choking fit, he was asleep at the time; he suddenly started up in bed with a feeling of suffocation as if something was rising in his throat. The pupils were unequal, the right being much smaller than the left. On March 11th, there was dullness at both bases, and he was found to be expectorating blood-stained frothy mucus. On March 15th, a well-marked pleuritic rub was heard in the right axilla. On March 17th, there were signs

of pleuritic effusion on the right side. On March 20th, aspiration was performed and 227 cubic centimetres of clear serous fluid were withdrawn. On April 3rd, he had several attacks of dyspnoea. On April 7th, he felt worse. He was still bringing up the blood-stained sputum. On April 9th, Cheyne-Stokes respiration was noted. On April 14th, he was much weaker, thinner and paler. He gradually became worse and died at 1 a.m. on the 15th.

*Post-mortem* 96, 1887.—The post-mortem was made by Dr. Pitt fourteen hours after death. The body was rather wasted; there was very little œdema. There was about half a litre of pus in the right pleural cavity, and there was a corresponding amount of compression of the right lower lobe. The pericardium was universally adherent but was not much thicker than normal; over the portion adherent to the right ventricle was a small, thick calcareous mass, for which no cause was obvious. The myocardium was fatty and mottled in colour on both sides. Both ventricles were dilated, especially the right; the left auricle was greatly dilated. The left ventricular wall measured 16 millimetres. There was chronic thickening of the endocardium on the left side. The mitral valve was stenosed; it measured 7.6 centimetres. The aorta was dilated and the valves were thickened and doubtfully competent. The tricuspid valve was thickened and it measured 14.3 centimetres. *The pulmonary artery looked very large, the valves measured 8.9 centimetres and were normal in appearance. The pulmonary branches were greatly dilated and the coat showed irregular cracks in the intima, which were blood stained.* These appeared to be due to over distension; there was no atheroma. There were numerous calcareous and fibroid patches in the aorta, especially in the first part. The liver weighed 1758 grammes; it was slightly nutmegged. The spleen weighed 255 grammes; it was tough. The kidneys weighed 312 grammes; they were tough and congested.

CASE 10.—N. H., an unmarried woman, æt. 23, came to my Out-patients for dyspnoea on October 29th, 1900. She had been troubled with shortness of breath for several years, getting it if she walked quickly and when she went upstairs. Five or six years ago she says that one day when coughing she burst a blood-vessel and brought up a quantity of blood. She states also that she has frequently brought up blood since, especially after exertion of any kind. Her menses are irregular. In June last she suffered from "quinsy." She gives no history of either rheumatism or scarlet fever. She says she has not lost flesh.

*Condition.*—Pulse 120, temperature 98.4°. There was marked dyspnoea, the *alæ nasæ* acting freely. There was no œdema of the legs and no ascites. The pulse was 120, a little irregular, and easily compressible. The cardiac impulse was seen in the sixth space about two centimetres outside the left nipple line; it was forcible in character and was also diffused. There was some epigastric pulsation. There was also a little bulging of the chest in the precordial area. A well-marked presystolic thrill was felt over the cardiac impulse. The cardiac dulness was increased (*vide diagram*). At the cardiac impulse a loud rumbling presystolic murmur was heard running up to and being continuous with a loud accentuated and prolonged first sound, which was followed by a blowing systolic murmur. *In the second and third spaces midway between the left border of the sternum and the left nipple line a soft, blowing, early diastolic murmur was heard.* The

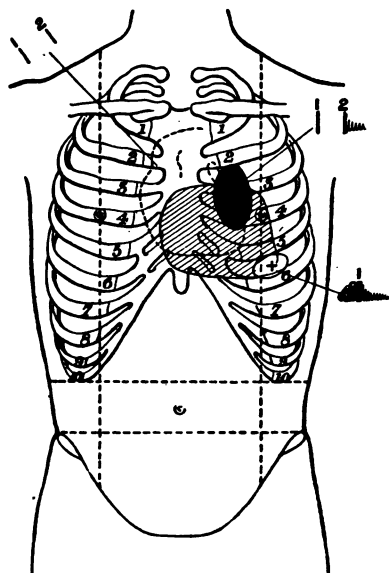


Fig. 6.

aortic second sound was sharp, clear and accentuated. She has been seen on several occasions and the diastolic bruit has always been present in the position described.

**CASE 11.**—Susan H. M., *set.* 14, under the care of Dr. Pye-Smith (clerk B. W. Moss), admitted February 17th, 1898, for precordial pain and cough. No family history of rheumatism. She has never been a strong child; four years ago she was admitted under Dr. Pye-Smith for chorea, and two years ago she was under Dr. Taylor, for rheumatism. When under Dr. Pye-Smith a diagnosis of mitral stenosis and regurgitation was made, the bruits heard being systolic and mid-diastolic in rhythm. When under Dr. Taylor, presystolic and systolic bruits were heard in the mitral area, and a systolic bruit in the pulmonary area. For the last twelve months she has been losing flesh, and her appetite has been very capricious, and for ten months previous to admission her cough has been very bad, and she has been short of breath. She has also suffered from pain and swelling at the back of her knees. There has been no swelling of her feet or ankles; no hæmoptysis or hæmatemesis.

*Condition on admission.*—Temperature 100°, pulse 100. She was a pale, thin, weakly looking child and did not appear to be in pain. *Circulatory system.*—Pulse 100, regular, weak and compressible. There was slight bulging of the chest in the precordial area. The impulse was visible in the fifth space, it was diffused in character and extended outwards about 2 centimetres external to the left nipple line. A presystolic thrill was felt over the mitral area. The cardiac dulness commenced above at the third left costal cartilage and extended below to the sixth; to the right it was limited

by the left border of the sternum, and to the left by a line drawn from the third rib through the nipple to the impulse. At the apex a loud, rough, rumbling presystolic bruit was heard, running up to a loud, sharp first sound, which was immediately followed by a harsh blowing systolic murmur, which latter bruit could be traced outwards into the left axilla. The presystolic

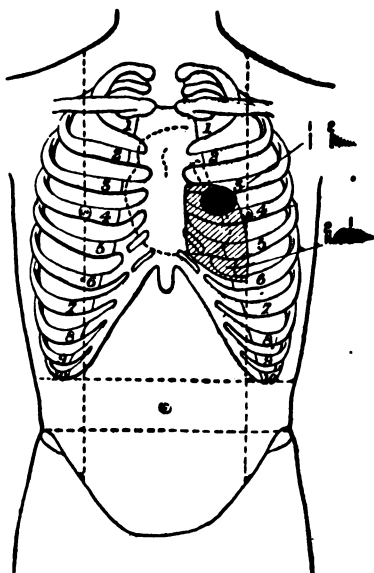


Fig. 7.

bruit followed immediately after a double second sound. *In the third left space, about 2 centimetres from the left border of the sternum, and 1.5 centimetres internal to the left nipple line, a faint, soft, blowing early diastolic murmur could be heard.* The aortic second sound was clear and distinct and there was no visible pulsation of the carotid arteries. No abnormal respiratory sounds. No ascites. Urine normal.

The patient left the hospital on March 2nd. The bruits at the apex altered from time to time, the presystolic being replaced by a mid diastolic bruit. The early diastolic murmur in the third left space, however, persisted, but it was not quite so well marked.

CASE 12.—Priscilla C., æt. 39, under the care of Dr. Shaw (clerk A. W. Talbot), Mary 19, and admitted on February 24th, 1898, for cough, dyspnoea and precordial pain. There was no family history of rheumatism. Her father suffered from gout and died at the age of eighty-one. She was married in 1888; she has no children. She has a cough every winter and expectorates a large quantity of phlegm. Two years ago, when hurrying to business, she had an attack of hæmoptysis. About November, 1897, she noticed dyspnoea on exertion. About a month previous to admission she began to suffer from precordial pain; fourteen days later the pain became

much worse, and it was so intense that it made her cry out, and as it gradually became still worse she came up to the hospital and was admitted on February 24th.

*Condition on admission.*—Temperature 97·6°, pulse 136, respiration 44; much cyanosed, veins in the neck pulsating. February 28th. *Circulatory system.*—Pulse 136, irregular, intermittent and compressible. Cardiac impulse visible in the fifth left space 2·5 centimetres outside the left nipple line, diffused and irregular; no thrill felt. Cardiac dulness is limited above by the third rib, to the right by the mid sternal line, below by the fifth space, and to the left by a line drawn downwards and outwards starting above just inside the left nipple line. On auscultation at the cardiac impulse the first sound was very loud, sharp, and slapping in character; it was preceded by a short, loud, rumbling presystolic murmur. The second sound in this position was loud and was followed by a soft, early, diastolic murmur. Just outside the impulse the first sound was loud and sharp in character and the early diastolic murmur was also audible. In the second and third left spaces midway between the left border of the sternum and the left nipple line a soft, early, diastolic murmur, which appeared to be different in its character to the above mentioned early diastolic murmur audible at the impulse, was heard. No bruits were heard in the aortic area. A few râles were heard at the bases of the lungs behind. On March 2nd, the presystolic murmur could not be heard. The early diastolic murmur in the pulmonary area was, if anything, more distinct.

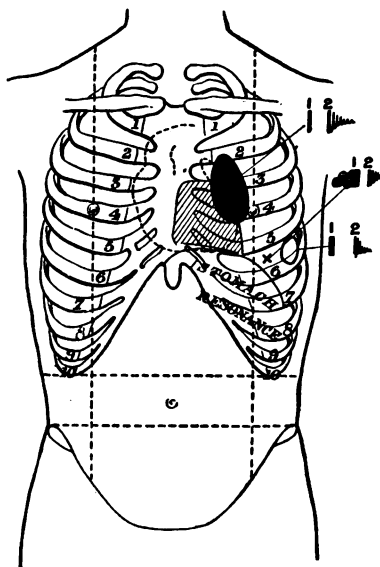


Fig. 8.

CASE 13.—Emily K., æt. 8, under the care of Dr. Goodhart (clerk H. E. C. Fox), admitted October 9th, 1897, for pain in the chest and shoulders. No family history of rheumatism or chorea. She has always been a delicate child, and when quite young suffered from an attack of congestion of the lungs following measles. Six years ago she had a bad attack of scarlet fever, which left her in a very weak state; since then she has suffered at times from fainting fits. Six weeks before admission the mother first noticed symptoms of chorea, viz., involuntary twitchings of the muscles of the hands and face.

*Condition on admission.*—Pulse 120, respiration 36, temperature 101.2°. No signs of chorea. The cardiac impulse was visible in the fourth space in the nipple line. A presystolic thrill was felt over the mitral area. For cardiac dulness *vide* diagram. On auscultation, a presystolic bruit was heard,

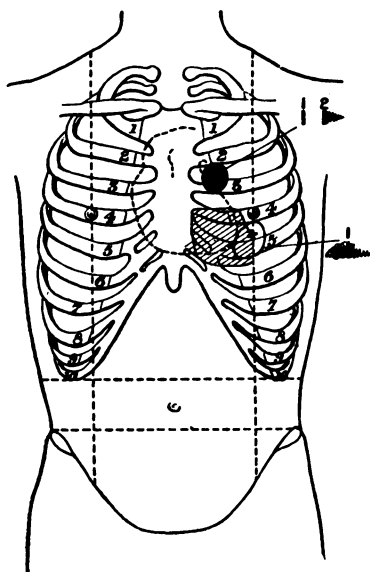


Fig. 9.

running up to a loud accentuated first sound, which was immediately followed by a loud blowing systolic murmur, traceable outwards into the left axilla. On October 21st, a loud and accentuated second sound followed by a soft, blowing, early diastolic murmur was heard over the second left space just outside the left border of the sternum. The aortic second sound was sharp and clear and no bruit could be heard accompanying or following it. On November 4th, the second sound in the pulmonary area was loud and slapping in character and there was immediately following it a well-marked blowing diastolic murmur. On January 3rd, 1897, she was discharged. She was very much better and the pulmonary diastolic bruit was not audible.



CASE 14.—M. H., *æt.* 12, a schoolgirl, was admitted 23rd February, 1897, under the care of Dr. Taylor (clinical clerks E. B. Dowsett and D. Munro) for dyspnoea and swelling of feet. There was no history of any previous illness, no scarlet fever, no rheumatism and no chorea, though the patient had always been weakly. The family history was good. She had generally had a winter cough and sometimes had had pain in her shoulder. The present illness had commenced two years ago, after a fall from a ladder, when she injured her left knee. She had limped since the accident, had lost flesh and had complained of shortness of breath, palpitation and pain at her heart. About the 3rd February she had become much worse had an aggravating dry cough, her ankles had swollen and she had been obliged to sit up at night for breath. Latterly the pain at her heart had been worse, and her face had become purplish. There had been no vomiting and no hæmoptysis.

*Condition on admission.*—Temperature 98°, pulse 100, respiration 24. She was thin and anæmic. Both knees were large but showed no signs of injury. The venules on the face were injected and prominent. *Circulatory system.*—Pulse regular, full, dicrotic, easily compressible. There was marked bulging of the precordia. The cardiac impulse was very diffuse, most marked in the sixth space 2·5 centimetres external to the nipple, but also seen in the seventh space 4 centimetres external to the nipple. Some sucking in of the intercostal spaces during systole was noticed. The cardiac dullness was limited above by the second left space, internally by the mid-line of the sternum, and it extended outwards to a point two centimetres external to the nipple, but there was partial dullness over a larger area (*vide diagram*). A well marked presystolic thrill

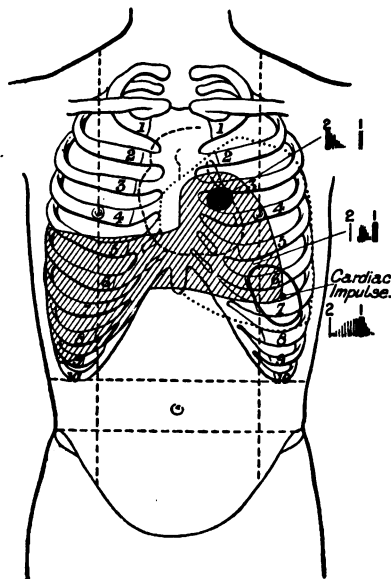


Fig. 10.

was felt over the cardiac impulse. A loud rumbling presystolic bruit with a loud first sound, was heard at the apex, followed by a faint

blowing systolic murmur traceable outwards. In the third left space, near the sternum, the first sound was reduplicated, the second sound was loud and was followed by a faint blowing diastolic murmur. Respiration was laboured in the supine position, but was easy and regular when the patient was propped up with pillows. There was a troublesome cough but no expectoration. There was some dullness at the left base as high as the angle of the scapula and some broncophony along the vertebral border of the left scapula. Loud rhonchi were heard over the upper half of the right chest and some very fine crackling râles were audible on both sides on deep inspiration. The urine was acid, sp. gr. 1018, and contained a trace of albumen. The abdomen appeared normal. Mitral stenosis and regurgitation was diagnosed. On March 1st, a soft systolic bruit was heard over the ensiform cartilage. The reduplication of the first sound in the third left space was more marked. On March 2nd, the diastolic murmur in the third left space was more marked but very localised. On March 4th, a soft blowing systolic murmur was heard in the second right space. On March 12th, the diastolic murmur in the third left space disappeared and was not heard again during the remainder of patient's stay in the hospital.

Patient continued to improve, and when she was discharged on March 23rd, the systolic bruit at the apex was loud and traceable into the axilla and behind. There was also a well-marked presystolic bruit (? at apex). The pulse was 120, regular and soft, and not nearly so dirotic in character. There was still some dullness at the left base over which crackling râles were heard, and a loud rhonchus was audible at the left apex posteriorly.

She was readmitted April 1st, for herpes zoster of the right buttock and outer side and front of right thigh. The condition of the heart was about the same, but no diastolic bruit was heard in the third left space. She left the hospital again on April 17th.

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CASE 15.—J. A., æt. 15 years, schoolboy, was admitted February 1st, 1897, under the care of Dr. Goodhart (clinical clerk R. H. J. Swan) for cough and shortness of breath. His illness commenced about 20th December, 1896, with an "attack of shivering," and he commenced coughing. The coughing continued and a few weeks later his legs began to swell. His face then swelled slightly, but afterwards the swelling went down. During the second week of the coughing he spat up some reddish brown blood. He had dyspnoea on exertion, but no palpitation. There was a history of chorea four years ago, for which he was treated at out-patients, and of pains in the joints during the last two years. His mother was suffering from rheumatic fever at the time of admission, but otherwise the family history was good.

*Condition on admission.*—Temperature 98.4°, pulse 134, respiration 36. Patient was well nourished, his cheeks were flushed and he was slightly jaundiced. There were slight choreic movements of the upper lip. The legs and ankles were cedematous. *Circulatory system.*—Pulse full, irregular, easily compressible and markedly dirotic. The cardiac impulse could not be seen but was felt in the fifth space 12 millimetres internal to the nipple line. There was a presystolic thrill. A rumbling presystolic bruit was heard at the apex, a soft, blowing systolic murmur traceable outwards to the axilla, and these were followed by a sharp second sound. Above, the cardiac dullness

reached to the lower border of the third rib, internally it was limited by the mid sternal line, while it extended outwards for 11 millimetres beyond the nipple line. *Respiratory system*.—Impaired resonance posteriorly at both bases, where there were some râles and occasionally rhonchi heard. *Alimentary system*.—Tongue furred, abdomen rigid, liver dulness extended from sixth rib to four centimetres below the costal margin in the nipple line. The front of the abdomen gave a tympanitic note on percussion. In the flanks the resonance was impaired. No thrill could be obtained. *Urine*.—Acid, sp. gr. 1012. Albumen absent. On February 4th, cardiac impulse seen, but no thrill felt. Râles, rhonchi and diminished resonance at the right base posteriorly. *A faint soft diastolic murmur was heard in the second left space and could be traced downwards and outwards for about three centimeters (Dr. Goodhart).* On February 6th, the cedema of the legs had disappeared. The abdominal wall was still rather resistant. On February 9th, urine acid, sp. gr. 1020, a trace of albumen present. On February 12th, rhonchi heard all over chest. On February 15th, a reduplicated second sound was heard at the base. On February 16th, urine, sp. gr. 1024, acid; small amount of albumen present. On February 17th, heart-sounds very irregular. Presystolic and systolic bruits at the apex and a reduplicated second sound at the base, heard best at the junction of the third left cartilage with the sternum. Occasionally the sounds presented a triple character. Venesection was proposed but not carried out. On February 18th, a systolic bruit was heard at the right of the lower end of the sternum. The heart sounds were extremely irregular, the beats often occurring in groups of three, and occasionally a beat was missed. On February 20th, the temperature was 100·6°, pulse 120, respiration 60. On February 22nd, an increase in the area of cardiac dulness was noted. Above it extended to the lower border of the second costal cartilage, externally to 13 millimetres beyond the nipple and internally it was limited by the right margin of the sternum. Systolic and presystolic bruits were heard at the apex, but at the base the sounds were normal. Dulness and deficient entry of air at the left base posteriorly, was noted. On February 23rd, great dyspnoea and much coughing. Rising temperature. Temperature (6 a.m.) 100·6°. On February 26th, very severe dyspnoea. Systolic bruits at the apex and over tricuspid area. Slight cedema of the feet and legs (2 p.m.). Respiration 76, pulse 138. Paracentesis thoracis 227 cubic centimetres of greenish fluid withdrawn from the left chest. Sp. gr. 1012, containing twenty-eight parts per thousand of albumen, 0·1 per cent. of urea. The fluid coagulated on cooling. On March 27th, he was still dyspnoeic (2 p.m., respiration 68, pulse 100). The air entry was better. Presystolic bruit at apex and systolic bruit at the lower part of the right side of the sternum. On March 1st, he was much better. Cardiac dulness less extensive; no tricuspid bruit. The sounds at the base were normal. On March 4th, presystolic and systolic bruits at the apex, soft systolic over the tricuspid area. Loud second sounds in the pulmonary area. On March 5th, soft systolic murmurs heard over the aortic and pulmonary areas. On March 10th, systolic and mid-diastolic bruits at the apex. Systolic murmur in the tricuspid area. Second sound accentuated over the pulmonary area and the first sound of a loud "scratching" character. General condition improving. On March 17th, cardiac impulse in the fifth space 13 millimetres internal to the nipple. Dulness extends 2 centimetres to the right of the sternum. Systolic and mid-diastolic murmurs at the apex;

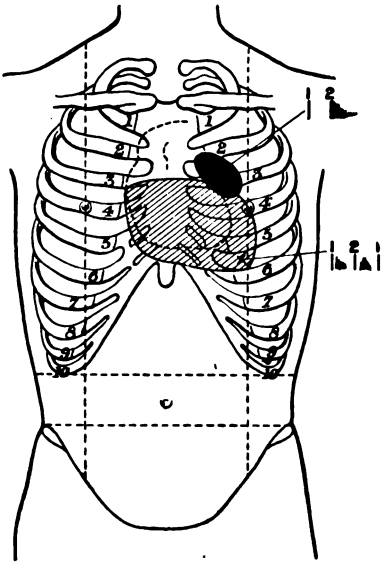


Fig. 11.

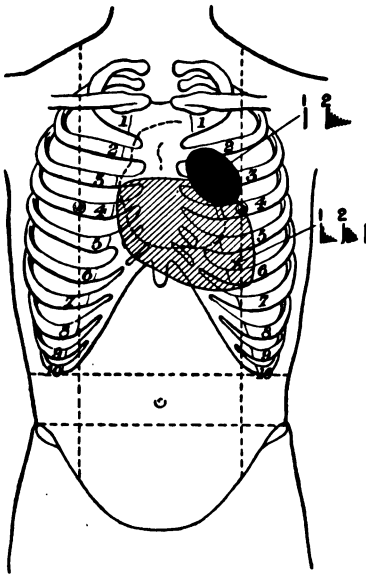


Fig. 12.

none over the tricuspid area. Both sounds loud and the second thickened over the pulmonary area. *At the junction of the second left costal cartilage with the sternum and traceable downwards and outwards half-way towards the left nipple a short, soft diastolic murmur was heard.* On March 18th, the pulmonary diastolic bruit was much better heard over the same area. A triple sound occasionally heard as if the first sound was reduplicated, but no reduplication can be heard at the apex. Patient better and getting up after dinner. On March 20th the pulmonary diastolic bruit was more distinct. On March 22nd the pulmonary diastolic bruit was traceable slightly upwards and outwards towards the left shoulder as well as towards the left nipple. Systolic bruit again heard in the tricuspid area. Bruits at the apex as before. The pulmonary diastolic murmur was not heard on the 24th, but was noted again having the same character and distribution on March 26th and 29th (*vide diagram*). After this it was not again heard. Patient went out relieved on April 8th.

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CASE 16.—E. B., æt. 19, general servant, was admitted 15th March, 1895, under the care of Dr. Shaw (clinical clerk R. Mathias) for heart disease. She had been in St. Thomas's Hospital nine years before (1886) for chorea. She suffered from breathlessness and cough in the winter of 1893-4, and had had influenza and "inflammation of the lungs" about Christmas, 1894. She had only followed her present occupation for three months; prior to that she had always lived at home. She had had influenza again three months before admission, and since then she had had a cough and had suffered from breathlessness, precordial pain and palpitation after meals, but her appetite had been good. Her legs and face had been swollen about March 1st—a fortnight before admission. She had not menstruated for three months prior to that she had been regular. Her mother had suffered from rheumatism, but otherwise the family history was good.

*Condition on admission.*—Temperature 98·8°, pulse 74, respiration 20. She was short and well-nourished; there was no œdema. *Circulatory system.*—The impulse was diffuse and felt in the fifth space as far out as 3·5 centimetres beyond the left nipple line. There was a thrill. The cardiac dulness commenced above at the third rib and extended a few millimetres to the right of the sternum. Presystolic and systolic bruits were heard at the apex, and systolic bruits were heard in the pulmonary, aortic and tricuspid areas. In the pulmonary area the second sound was accentuated. The pulse was small, regular and compressible. The respiratory system was normal; there was no cough. She complained of a pain in her side but no abnormal physical signs were detected. The urine was acid, sp.gr. 1014, and contained no albumen. The abdomen appeared normal. On March 17th, in addition to the other bruits, *a diastolic murmur was noticed in the second and third left intercostal spaces* (Dr. Shaw) (*vide diagram*). *The same diastolic murmur*

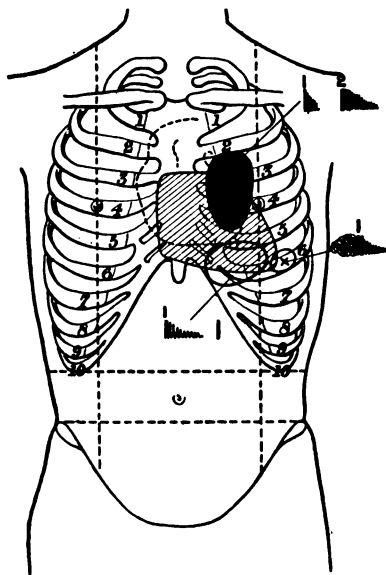


Fig. 13.

*was also heard March 20th (J. H. B.) On March 23rd it was not audible (Dr. Shaw) and it was not noted again. Patient's condition improved and she went out on April 4th.*

# TWO NÆGELE PELTS.

(WITH FOUR PLATES.)

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By J. H. TARGETT, M.S.

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THE rarity of this form of pelvic contraction is sufficient justification for recording all examples of it, although there may be little to add to the anatomical descriptions which have already been given so fully by Nægele and many subsequent writers. The first specimen (Pelvis A.) was removed from a case which died in the hospital, and some clinical notes of this case have been preserved; however, no exact diagnosis of the kind of pelvic deformity which existed was made during life, as the patient was extremely ill from obstructed labour and the foetal head was impacted at the outlet. From the testimony of friends it may be concluded that she presented no obvious deformity and that her gait was quite normal.

The second specimen (Pelvis B.) has been preserved in the hospital museum for more than fifty years, but there are no records of any kind concerning it. As it is an excellent example of the deformity and exhibits some peculiarities, a full description of the pelvis has been prepared. It is impossible to estimate the frequency of the Nægele pelvis. In the Chrobak Klinik, among 50,000 labours the Nægele pelvis was only once found, though contracted pelvis of other varieties occurred in 3·8 per cent. of the cases. In 38,000 labours at the Royal Maternity of London

no case of Nægele pelvis was discovered. And in the Guy's Hospital maternity records, which have been kept since 1833, there is no mention of any case similar to the one related below.

On the other hand, out of a total of 17,000 labours in various American hospitals, Professor Lusk found no less than five cases of Nægele pelvis recorded.

The best collection of cases of difficult labour from this form of pelvic contraction has been made by Dr. Tchérépakchine, of Paris, in a monograph published in 1893. He records some seventy cases, of which fifty-four are accompanied by clinical histories, and from these figures we may gather some idea of the fearful mortality which has attended such labours in the past. It will be admitted that as most of the cases occurred before the days of antiseptic midwifery, a similar mortality would not obtain in the future; but the frequency of severe complications, such as rupture of the uterus, and the probability that the deformity will not be discovered until difficulties have arisen, combine to make this variety of pelvic contraction one of peculiar interest and importance.

Of the fifty-four cases above referred to, no less than forty-four (80 per cent.) have left their pelves to various museums as the result of gestation. Of these forty-four fatal cases, six died undelivered, thirteen had rupture of the uterus and vagina and died of hæmorrhage or peritonitis, and the remainder succumbed to puerperal septicæmia in one form or another.

The ten women who survived their confinements were delivered by version, forceps, or craniotomy, the operations being attended with considerable difficulty. The majority of them were multiparæ, and had experienced similar difficulties with their previous confinements.

If the results of the previous labours of the whole series of cases (fifty-four in number) be tabulated, we have a list of 112 confinements, and deducting the six cases which died undelivered, as well as seven others, of which the records are incomplete, the modes of termination of 100 labours (including the case recorded below) may be thus stated:—



(a). Labours ending naturally at term ...	15	(7 cases)
(b). Labour ending naturally, but before term ... ..	1	(1 " )
(c). Labours induced and ending naturally	7	(3 " )
(d). Labours induced, but requiring operations ... ..	12	(3 " )
(e). Labours at term ended by forceps ...	21	(17 " )
(f). Labours at term ended by version ...	6	(5 " )
(g). Labours at term requiring perforation, craniotomy, &c. ... ..	32	(21 " )
(h). Labours at term—Cæsarian section ...	4	(4 " )
(i). Labours which presented extreme difficulty ... ..	2	(2 " )
<hr/>		
100		

It should be noted that with the fifteen labours ending spontaneously at term, only seven women were concerned; for one case was delivered naturally five times, another four times, and another twice. Similarly of the three patients in whom labour was induced and ended naturally, one was so delivered five times. The very small proportion of cases in which version at term was successful is significant, and the explanation is not difficult. In addition to the obstruction at the pelvic brim resulting from the ankylosis and oblique contraction, the typical Nægele pelvis exhibits a marked degree of inversion of the ischial spine and tuberosity on the affected side. In consequence the pelvic cavity becomes funnel-shaped and the obstruction is progressively increased in passing from the brim to the outlet. Both the specimens here described exhibit a high degree of contraction at the pelvic outlet, and owing to the excessive inversion of the ischial spines the sacrum is entirely prevented from taking any share in the formation of the circle which is included between these spines and the walls of the pelvis, on a level with the apex of the pubic arch. Indeed, the outline of the plane of the cavity at this level is in shape like a figure of 8, with unequal loops. Of these loops the posterior, which lies between the ischial spines and the front of the sacrum, is the smaller, and

is much flattened from before backwards and obliquely, like the brim; the anterior loop, on the other hand, is considerably larger and rounded. From these considerations it follows that nothing is gained by podalic version, and if the head is already in the pelvic cavity there is the additional risk of injury to the uterus from the operation itself. Cæsarian section at term is now the most suitable method of dealing with these cases provided the child is alive, and the patient must be sterilised at the same time. But if repeated attempts at delivery with forceps have been made, and risks of injury to the mother and child have thus been incurred, it will probably be safer to perform craniotomy. On a subsequent occasion, when the true characters and measurements of the pelvis are known, the choice would lie between induction of premature labour or Cæsarian section. It is particularly important that the distances between the two ischial spines, or the tuberosities, should be measured, and this would be most satisfactorily done under anæsthesia.

The following are the most important measurements of the pelvis, which should be taken on each side and compared:—

1. From one anterior superior iliac spine to the opposite posterior superior spine.
2. From both anterior superior spines to the fifth lumbar spine.
3. From one posterior superior spine to the opposite tuber ischii.
4. From one posterior superior spine to the opposite trochanter major.
5. From both posterior superior spines to the fifth lumbar spine.
6. From the lower edge of the symphysis pubis to both posterior superior spines.
7. From the lower edge of the symphysis pubis to both ischiatic spines.
8. From both ischiatic spines to the nearest point on the edge of the sacrum.
9. From the tip of the sacrum to both tubera ischii.

10. From the centre of the promontory of the sacrum to both ilio-pectineal eminences (sacro-cotyloid diameter).

In addition to these, the majority of which are peculiar to the Nægele pelvis, there are the usual external measurements—the interspinous, the intercrystal, and the external conjugate—and the various diameters of the pelvic brim, cavity and outlet. The importance of the inter-ischial and transverse diameter of the outlet has been already mentioned.

#### PELVIS A.

From a case of difficult labour due to an obliquely contracted (Nægele) pelvis. Delivery by cephalotripsy after repeated applications of the forceps. Death from acute septicæmia and peritonitis due to rupture of the uterus.

#### CLINICAL HISTORY.

Charlotte F., aged 24, primipara, was admitted to Guy's Hospital on Tuesday, February 14th, 1899. Labour began on Saturday, February 11th, and continued until Monday, February 13th, at 6 p.m., when forceps were tried, but unsuccessfully. At 9 p.m. the same evening an anæsthetic was given and forceps were again applied. After repeated attempts at extraction it was decided to recommend the removal of the patient to Guy's Hospital; but the friends were at first unwilling, and it was not until Tuesday, February 14th, at 6 p.m., that the patient was admitted. She had then been in labour some eighty-four hours.

*On admission.*—The patient was very drowsy, with eyes sunken, and pupils contracted as though under the influence of opium. Temperature 98·6°, pulse 140–150, small and running. She was evidently in a state of profound septicæmia. The abdomen was extremely tender to touch, and the uterus was closely contracted round the child, though no retraction-ring was visible. The uterus felt hard but varied in that respect, and was not therefore in a condition of absolute tonic contraction. The foetal heart could not be heard, and owing to the extreme tenderness, no further abdominal examination was made. By the vagina a vertex presentation was felt, covered with a large caput, which

1107A

was only one inch and a half from the vulva. There was not much œdema of the vulva, but numerous lacerations could be felt in the walls of the vagina. The patient was immediately anæsthetised, the external parts were shaved and thoroughly cleaned, and then craniotomy was performed in the dorsal position without making any trial of forceps. The site of perforation was afterwards found to be just behind the right parietal eminence, so that the presentation had been left occipito-anterior. The brain matter which escaped had a distinctly foul odour. The cephalotribe was applied in the transverse diameter of the pelvis, and the grip obtained was so good that it was unnecessary to change the position of the instrument. The head was slowly delivered in the antero-posterior diameter after a good deal of traction, the difficulty being attributed chiefly to the undilated condition of the soft parts and perineum, as the peculiar nature of the pelvic contraction was not suspected. The posterior arm was then withdrawn, and the rest of the body followed without much difficulty. The perineum, however, was ruptured for about an inch, and required three sutures for its repair. During this operation an injection of strychnine and atropine was given, and after the patient had been returned to bed, a coffee enema was administered. 11 p.m.: the pupils were still contracted, the face was suffused, the lips were livid, and the breathing was extremely rapid and shallow. Respiration 50-60, pulse 170, temperature 99.6°. The abdomen was flatulent and distended, but less tender, and there was no sickness. As the condition clearly indicated acute septicæmia of the worst type, 20 cubic centimetres of antistreptococcic serum were injected into the loin.

February 15th: In the early morning sickness began and continued for several hours. The pulse and respiration were still very rapid, but the temperature was only 98°. 10.30 a.m.: the bowels acted freely, but the abdomen had become more distended. Pulse 160, respiration 52, temperature 99. A little later the pulse was uncountable, the breathing more rapid, and the abdomen very tender. Death occurred at 4 p.m., or twenty-two hours after admission, from acute septicæmia and peritonitis.

1891

The autopsy revealed a rupture of the lower segment of the uterus extending into the vagina, and opening the peritoneum in Douglas' pouch.

The previous history of the patient was to the effect that she was in excellent health up till the time of her confinement, that she had never been seriously ill, and that no limp, or lameness, or curvature of the spine had been noticed by her friends. She was a strong active young woman, and a good walker.

The general characters of the dried pelvis are exhibited in the photographs. (Plates 1 and 2). There is a deficiency in the development of the right ala of the sacrum, and of the adjoining part of the right ilium, and the right sacro-iliac joint is partially ankylosed. The right iliac fossa is smaller and much less concave than the left. The epiphyses for the iliac crests are not yet united. In other respects the right os innominatum is as well developed as the left.

*Sacrum.*—The base of this bone shows a congenital abnormality in its formation, which is quite distinct from the ill-development of the right ala. This abnormality consists in the production of two long costal processes for the first sacral vertebra, which are quite separate from the alæ. In fact, the first sacral vertebra very closely resembles the last lumbar; it has a long narrow spinous process; the laminæ are distinct and not ossified to the succeeding laminæ; the superior and inferior articular processes are well formed, and the costal processes are in a line with those of the last lumbar vertebra. That it is not an additional (sixth) lumbar is proved by its ankylosis to the remainder of the sacrum, which is composed of only four vertebræ, and by the position of the first sacral foramina which run between the alæ and the above mentioned costal processes. These foramina are very small and slit-like both in front and behind, as though compressed from above downwards. The body of the first sacral is much less deep than usual, and the position of the first intervertebral ridge is obscured by an irregular ossific deposit upon the anterior aspect of the sacrum. But the interval between the first and second centra can be recognised posteriorly, and the depth of the first vertebra behind is found to be under 2 centimetres.

Before leaving the first sacral costal processes, it should be noted that the right one is somewhat longer than the left, and is directed more strongly backwards in consequence of the increased traction on the right posterior sacro-iliac ligaments, many bundles of which are inserted into it. The extremity of the right costal process is in close contact with the ilium, but does not articulate with it; on the left side, owing to the greater width of the ala, the end of the costal process is some distance from the ilium.

From the foregoing description of the first sacral costal processes it will be understood that they do not enter into the formation of the alæ, and that the promontory is therefore elevated above the base of the sacrum, or more accurately above the plane of the pelvic brim, by the depth of the first sacral vertebræ, which is roughly 1.5 centimetres.

The alæ are formed chiefly from the costal processes of the second sacral vertebræ, and the succeeding vertebræ have a normal arrangement. The right ala is considerably smaller in all directions than its fellow. From the median-line of the sacrum to the sacro-iliac joint is 3 centimetres on the right side and 5 centimetres on the left. Measurements of the alæ from before backwards and from above downwards also show differences in favour of the left side. There is an irregular ossific deposit on the front of the sacrum which affects the anterior surface of the first four bodies. It is, however, limited to the bodies, and does not extend beyond the line of the sacral foramina. This limitation is of importance, for it excludes the possibility of attributing the ankylosis of the right sacro-iliac joint to any inflammatory lesion in the articulation. The bony surfaces in the vicinity of both joints are quite smooth, and free from any indication of disease. The cause of the ossific deposit on the bodies is not obvious. The character of the new bone indicates an osteoplastic process from periosteal irritation without evidence of caries or necrosis. There is no similar change on the posterior aspect of the sacrum, nor indeed in any other part of the pelvis. The most striking features on the back of the sacrum are the presence of a separate and well developed first sacral spinous process, and the diminished width of the right half of the bone. From the

median-line behind to the level of the sacro-iliac joints is 3 centimetres on the right side, and 4·5 centimetres on the left. The outlines of the inferior articular processes of the first vertebra are very distinct, but they are completely ankylosed to the superior processes of the second vertebra.

*Ossa innominata.*—The right hip bone differs from the left chiefly in the smaller size of its iliac portion and the diminished curvature of the whole bone, especially along the ilio-pectineal line. The right iliac fossa is much less concave than the left, and measures 10·5 centimetres by 8·5 centimetres, as against 12 centimetres by 9 centimetres on the opposite side. The posterior extremity of the right ilium, where it enters into the sacro-iliac joint, is somewhat smaller than its fellow, but the difference is not great. In this respect, the specimen now under consideration is in marked contrast with pelvis B, in which the corresponding part of the ilium is very ill-developed. The remaining parts of the right os innominatum show no deficiencies in development, and except for alterations in curvature and direction, the bones are alike on both sides.

*Sacro-iliac joints.*—There is complete bony ankylosis of the right joint along its anterior inferior and posterior aspects, and though the line of the articulation can be traced without much difficulty, yet the articulating edges are so completely welded as to form a perfectly smooth surface. On the superior aspect of the right joint, along the base of the sacrum, the line of the articulation is quite distinct, and the edges of the bones, though in very close apposition, are not ankylosed. In the recent state no movement could be obtained between the ilium and sacrum on the right side. On tracing the outline of the right joint, it will be seen that the ala exhibits above a very pronounced “bite” or triangular projection, which is firmly wedged into a corresponding depression in the ilium. The situation of this “bite” probably marks the ridge between the articular and non-articular surfaces of the right ala, but it is impossible to make sure of this without damaging the specimen. The lower limit of the right joint is nearly on a level with the left, but owing to the atrophy of the right posterior inferior iliac spine, and the increase in height of the great sacro-

sciatic notch, the area of the right articulation seems much smaller than it is in reality. The circumferences of the two joints, when carefully traced with a tape measure, showed a difference of little more than one centimetre in favour of the left side. At the back of the right joint it will be noticed that the fibres of the posterior sacro-iliac ligament run almost directly backwards and a little upwards, while on the opposite side they run outwards and backwards. If the lines of these bands be prolonged forwards they will meet in front of the sacrum, almost at a right angle. More striking still is the altered relation of these ligaments to the sacro-cotyloid beam, or that thickened portion of the os innominatum which extends from the synchondrosis to the summit of the acetabulum. On the right side the angle formed between a line through the posterior sacro-iliac ligaments and the long axis of the adjacent sacro-cotyloid beam is  $30^{\circ}$ ; on the opposite side it measures  $75.5^{\circ}$ . The mechanical advantage thus gained on the left side goes far to explain the greatly increased curvature of the left side of the pelvic brim and its diminution or flattening on the ankylosed side. Moreover, the right ala has sunk somewhat forwards into the pelvis so that there is a backward projection of the right ilium behind the limit of the synchondrosis, amounting to at least one centimetre in excess of that on the opposite side.

The condition of the sacro-sciatic notches will next engage attention. The right notch differs from the left in width and in the level of its summit, but the actual depth of the notch is the same on both sides. The width of the right notch is 4.5 centimetres, and of the left 6 centimetres; while the summit of the right notch is about 1 centimetre higher than the left—a difference which corresponds very closely with the increased backward and upward displacement of the right hip-bone, as described above. But not only is the right notch narrower than its fellow, there is also a marked inward displacement of the right ischium and its spine, whereby the sharp anterior edge of the right notch is carried inside the plane of the right margin of the sacrum. The corresponding edge of the left notch is in consequence thrust outwards, and if the distance of this edge from the median plane of the sacrum be taken it will be found



that this distance measures only 3 centimetres on the right side and 6·5 centimetres on the left. This inward thrust of the ischium and its spine on the ankylosed side is of very great importance, because it leads to a peculiar figure-of-8 contraction of the plane of the pelvic cavity at that level which practically corresponds with the plane of the outlet.

The measurements of this pelvis are as follows :—

1. Anterior interspinous diameter	...	...	22·5 centimetres.
2. Intercristal	"	...	24·5 "
3. External conjugate	"	...	18 "
4. True conjugate	"	...	9·5 "
5. Diagonal conjugate	"	...	11 "
6. Posterior interspinous	"	...	6·5 "
7. From right A.S.S. to left P.S.S.	...	...	19 "
From left A.S.S. to right P.S.S.	...	...	21·5 "
8. From right P.S.S. to left tuber ischii	...	...	18 "
From left P.S.S. to right tuber ischii	...	...	14 "
9. From right A.S.S. to 5th lumbar spine	...	...	15·5 "
From left A.S.S. to 5th lumbar spine	...	...	17·5 "
10. From right P.S.S. to lower border of symphysis	...	...	18 "
From left P.S.S. to lower border of symphysis	...	...	16 "
11. From right P.S.S. to posterior median line of sacrum	...	...	3 "
From left P.S.S. to posterior median line of sacrum	...	...	3·5 "
12. From right ischiatic spine to edge of sacrum (nearest point)	...	...	4 "
From left ischiatic spine to edge of sacrum (nearest point)	...	...	6 "
13. From right tuber ischii to tip of sacrum	...	...	8 "
From left tuber ischii to tip of sacrum	...	...	10 "
14. From centre of sacral promontory to right ilio-pectineal eminence	...	...	6·5 "
From centre of sacral promontory to left ilio-pectineal eminence	...	...	10·5 "

- |   |     |             |     |     |              |
|---|-----|-------------|-----|-----|--------------|
| 15. From promontory to right (ankylosed)        |     |             |     |     |              |
| sacro-iliac joint                               | ... | ...         | ... | 3.5 | centimetres. |
| From promontory to left sacro-iliac             |     |             |     |     |              |
| joint   | ... | ...         | ... | 5   | "            |
| 16. The antero-posterior diameter of the sacral |     |             |     |     |              |
| promontory passes through the right             |     |             |     |     |              |
| body of the pubes                               | 2.5 | centimetres | to  |     |              |
| the right of the symphysis.                     |     |             |     |     |              |
| 17. Transverse diameter of pelvic brim          | ... | 11.5        |     | "   |              |
| 18. Right oblique diameter of brim              | ... | 12.5        |     | "   |              |
| Left oblique diameter of brim                   | ... | 9.5         |     | "   |              |
| 19. Antero-posterior diameter of outlet         | ... | 11.5        |     | "   |              |
| Transverse diameter of outlet                   | ... | 9.5         |     | "   |              |
| Between spines of ischia                        | ... | 8           |     | "   |              |
| 20. From lower edge of symphysis to right       |     |             |     |     |              |
| ischial spine                                   | ... | 9           |     | "   |              |
| From lower edge of symphysis to left            |     |             |     |     |              |
| ischial spine                                   | ... | 7.5         |     | "   |              |

#### PELVIS B.

The general characters of this pelvis are well shown in the accompanying photographs (Plates 3 and 4). There is complete osseous ankylosis of the right sacro-iliac articulation, and marked deficiency in the development of the right ala of the sacrum and the right ilium. There is no evidence of arrested growth in any part of the ischium, pubes, or acetabulum, and except for such changes as result from alterations in the lines of pressure there is nothing abnormal in these parts.

*The sacrum.*—The right ala of the sacrum is much smaller than the left, and its width is especially deficient. From the centre of the promontory to the sacro-iliac joint is 4 centimetres on the right side and 6.5 on the left. The direction of the right ala is also much more horizontally outwards than the left. The first right anterior and posterior sacral foramina are somewhat smaller than those on the opposite side, but the remaining foramina in front and behind are not affected. Though the right sacro-iliac joint is completely ankylosed the line of the articulation is clearly indicated by a bony ridge, which shows that the costal processes

of the first two sacral vertebræ have entered into the formation of the right joint, while on the left side a part of the third vertebra contributes to the articulation. Viewed from the front there is a very striking difference in the depth of the sacral alæ, the right being much smaller than its fellow; but on closer inspection it is seen that this difference is due largely to deficiency in the development of the right ilium associated with increased depth of the right sacro-sciatic notch. The circumference of the right ala passing through the summit of the notch is 15 centimetres; on the left side it measures 19 centimetres. There are no important changes on the posterior aspect of the sacrum except such as relate to the right sacro-iliac joint, which will be described with the ilium. The coccyx also is normal.

*The right ilium.*—As already stated, the deficiencies in the development of that part of the ilium which enters into the formation of the right sacro-iliac joint constitute the most striking feature in the specimen. The epiphysis for the crest of the ilium is wanting on both sides, which probably indicates that the patient was about twenty years of age. On the right side, that rough portion of the iliac crest which constitutes the tuberosity of the ilium is wanting, and the posterior extremity of the crest ends prominently about three-quarters of an inch above the level of the sacro-iliac joint. Hence there is a considerable difference in the lengths of the two iliac crests: the left crest is 24 centimetres in length, while the right one only measures 16 centimetres. But this prominent termination of the right crest cannot be regarded as the true posterior superior iliac spine, for there is a minute representative of this spine on a level with that of the opposite side. Hence the piece that is wanting includes the greater part of that thickened posterior extremity of the ilium which is marked off by the superior gluteal ridge, and gives attachments to the gluteus maximus externally, and to the posterior sacro-iliac and ilio-lumbar ligaments internally. Both posterior inferior iliac spines are on the same level—about the upper border of the third costal processes of the sacrum. In consequence of the above-mentioned deficiency in the right bone there is a very marked difference in the width or antero-posterior measurements

of the two iliac bones. Thus, the left ilium measures 14·5 centimetres horizontally backwards from its anterior inferior spine to the edge of its tuberosity a little above the posterior superior spine, while a line drawn similarly on the right side only measures 11 centimetres. There is also much diminution in the width of the great sacro-sciatic notch on the right side as compared with the left. Thus, the greatest width of the latter is 5 centimetres, but the right notch is only 3·5 centimetres, and would probably be less but for the inward displacement of the right ischiatic spine, which has carried the anterior edge of the notch with it. The degree of this inward displacement of the spine of the right ischium and anterior edge of the great sacro-sciatic notch is very considerable, but can hardly be expressed in figures. From the anterior median-line of the sacrum opposite the centre of the third body horizontally outwards to the anterior edge of each notch measures 4 centimetres on the right side and 8 centimeters on the left. When viewed from the front the tip of the right ischiatic spine is in the same plane as the row of right sacral foramina, while the left ischiatic spine is at least 3 centimetres outside the plane of the left sacral foramina. The first sacral foramina both in front and behind on the right side are somewhat smaller than their fellows, but there are no differences in the succeeding foramina from those of the opposite side. The apex of the great sacro-sciatic notch on the right side is on a level with the middle of the body of the second sacral vertebra, but on the left side it is fully 2 centimetres lower, in fact, not much above the middle of the third body. In spite of this difference in level the actual length of the great sacro-sciatic notch, as measured from its apex to the tip of the ischiatic spine, is practically the same on both sides. Hence the elevation of the right notch corresponds with the upward displacement of the whole right os innominatum, and may be taken as the index of that upward displacement. Hence, as both os innomina~~t~~<sup>a</sup> are of the same length, the right iliac crest is 2 centimetres higher than the left, and the same holds good for the levels of the tuber ischii of each side.

On looking at the pelvis as a whole, the altered shape of the pelvic brim is well seen. The superior edge of the sacrum or promontory is very high, being fully 2 centimetres above the plane of the brim. Tracing the pelvic brim round from the front of the first sacral vertebra the line passes first horizontally outwards to the right. Immediately beyond the first sacral foramen the line turns sharply forwards, almost at right angles to the anterior surface of the sacrum, and is continued on to the symphysis pubis as the ilio-pectineal line. From the ridge which marks the ankylosed sacro-iliac joint to a point corresponding with the right ilio-pectineal eminence this line runs very nearly straight forwards, but beyond this point it has a slight gentle curve up to the symphysis. On tracing the left side of the pelvic brim we note that the line makes a very large curve from the front of the sacrum along its left ala and the left ilio-pectineal line as far as the corresponding ilio-pectineal eminence, thence onwards to the symphysis pubis the curvature of the outline is much diminished. If the plane of the pelvic brim be divided into two chords by a line from the centre of the promontory to the symphysis pubis, the height of the right chord (ankylosed side) is 4.5 centimetres, but that of the left chord is 7 centimetres. The convexity of the right chord is 16 centimetres, and of the left 20 centimetres.

Beside the obliquity of the pelvic brim, the backward displacement of the right ilium must be noted. This may be expressed in figures by marking the levels of the anterior inferior iliac spines upon the antero-posterior diameter of the pelvic brim. A line drawn from the front of the right spine at right angles to this diameter is found to be 4 centimetres behind the line similarly drawn on the left side. The whole of the right os innominatum is displaced backwards and upwards in relation to the opposite bone, its iliac fossa is smaller and less concave, and the long axis of the bone from the highest part of the crest to the tuber ischii is straighter than its fellow. Passing through the pelvic cavity to the outlet, there is a progressive diminution in width which renders the cavity funnel-shaped, as well as a very marked inversion of the right ischium, and especially of its spine, whereby the cavity near the plane of the outlet is greatly constricted. The interval

between the tips of the ischial spines is only 6·5 centimetres, and the inward projection of these spines is such that the sacrum is quite outside the circle formed by the plane of the pelvic cavity at that level. The diameter of this circle is about 8 centimetres, or just over three inches, and this represents the available space for the passage of the foetal head.

This approximation of the ischial spines is of the greatest importance from an obstetric point of view, and although it may not be measurable clinically, yet in high degrees of deformity, as in this pelvis, the inversion of these ischial spines would be easily recognised on vaginal or rectal examination.

The following are measurements of the pelvis as a whole:—

1. Interspinous diameter ... 21·5 centimetres ( $8\frac{1}{2}$  inches)
2. Intercristal " ... 24 " ( $9\frac{1}{4}$  " )
3. External conjugate " ... 16·5 " ( $6\frac{1}{2}$  " )
4. True conjugate " ... 11·5 " ( $4\frac{1}{2}$  " )
5. Diagonal conjugate " ... 13 " ( $5\frac{1}{8}$  " )
6. From right A.S.S. to left P.S.S. ... = 19 centimetres.
- From left A.S.S. to right P.S.S. ... = 19 "

Owing to the deficiency at the back of the right ilium, the termination of this diameter on the right side cannot be accurately fixed; hence this measurement loses much of its value.

7. From right A.I.S. to middle of sacral promontory ... = 9 centimetres.
- From left A.I.S. to middle of sacral promontory ... = 12·5 "
8. From right P.S.S. to left tuber ischii ... = 16 "
- From left P.S.S. to right tuber ischii ... = 14 "
9. From right P.S.S. to posterior median line of sacrum ... = 2·5 "
- From left P.S.S. to posterior median line of sacrum ... = 4·5 "
10. From right ischiatic spine to edge of sacrum (nearest point) ... = 2·5 "
- From left ischiatic spine to edge of sacrum (nearest point) ... = 4 "

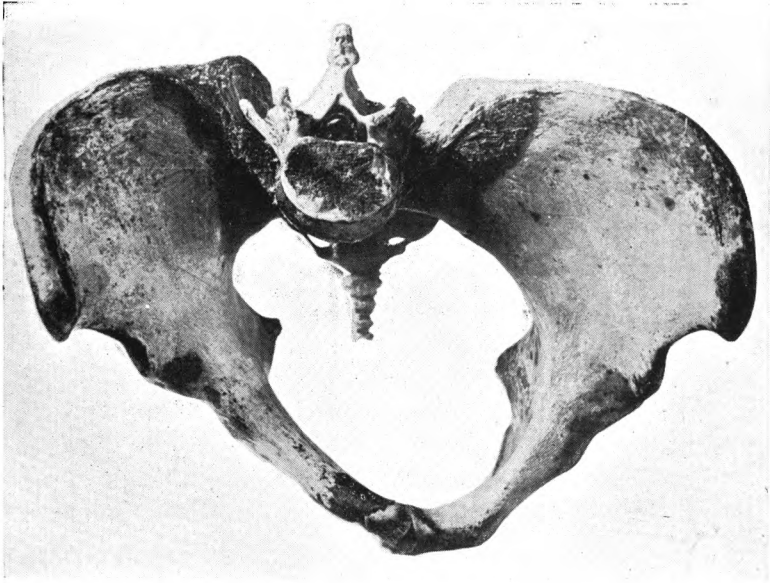
11. From right tuber ischii to tip of sacrum = 7 centimetres.  
     From left tuber ischii to tip of sacrum... = 9       "
12. From centre of sacral promontory to  
     right ilio-pectineal eminence ... = 7·5       "  
     From centre of sacral promontory to  
     left ilio-pectineal eminence... .. = 11       "
13. From centre of sacral promontory to  
     right (ankylosed) sacro-iliac joint ... = 4       "  
     From centre of sacral promontory to  
     left sacro-iliac joint ... .. = 6·5       "
14. The antero-posterior diameter of the sacral promontory  
     passes through the right body of the pubes 2·5 centi-  
     metres to right of the symphysis.
15. The right sacro-cotyloid diameter ... = 8 centimetres.  
     The left sacro-cotyloid diameter ... = 11       "
16. Right oblique diameter of brim... .. = 12·5       "  
     Left oblique diameter of brim ... .. = 10·5       "
17. The transverse diameter of brim ... = 11       "
18. The antero-posterior diameter of outlet = 11       "  
     The transverse diameter of outlet ... = 8       "  
     Between the spines of the ischia ... = 6·5       "
19. From the lower edge of the symphysis to both ischiatic  
     spines and to both posterior-superior iliac spines are  
     practically the same on the two sides, the difference in  
     favour of the right side not exceeding a quarter of an  
     inch.





*Two Nægle Pelves.*

PLATE 1.



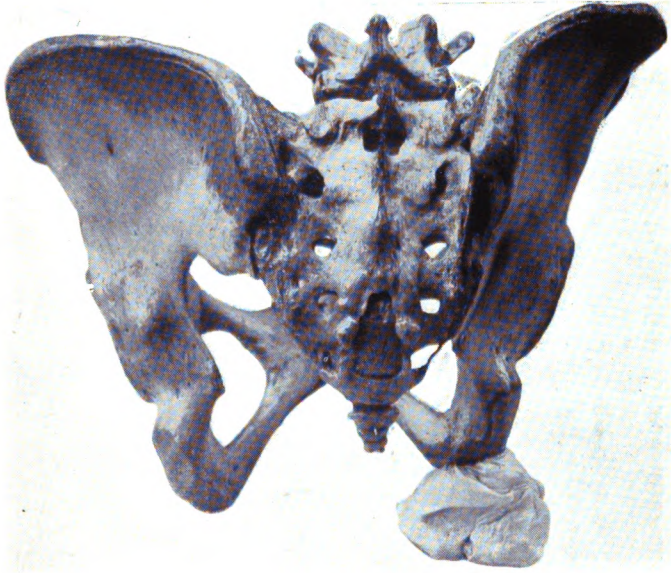
PELVIS A.—FIG. 1.

The pelvis (including the last lumbar vertebra) is viewed from above at right angles to the plane of the brim. The obliquely oval outline of the brim can be traced, and the figure-of-8 contraction of the cavity is well seen. Note the different directions of the posterior sacro-iliac ligaments as mentioned in the text.



*Two Nægele Pelves.*

PLATE 2.



PELVIS A.—FIG. 2.

The separate spine and arch of the first sacral vertebra are seen below the last lumbar. The first posterior sacral foramina are too small to be recognised. Note the diminution in width of the right half of the sacrum.

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*Two Nægele Pelves.*

PLATE 3.



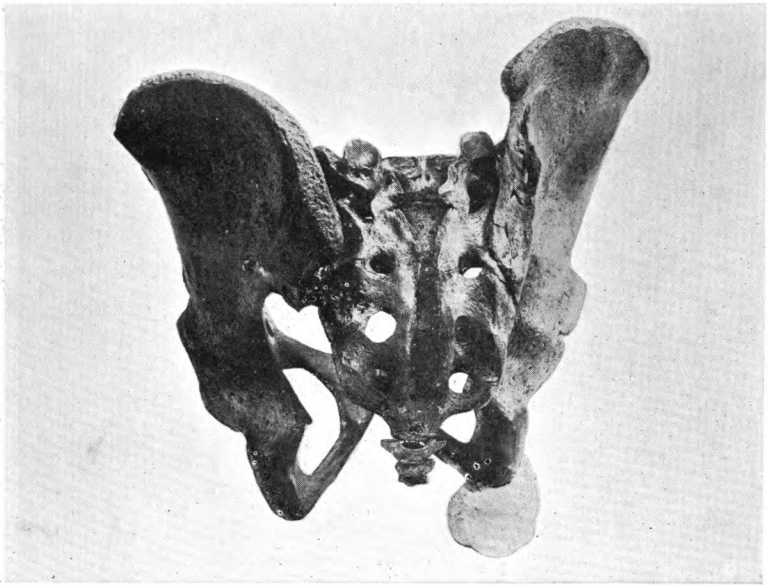
PELVIS B.--FIG. 1.

The inversion of the right ischial spine and the narrowness of the right sacro-sciatic notch are easily recognised. Note the difference in shape and elevation of the right ilium, and the flattening of the ilio-pectineal line on that side. The epiphyses for the iliac crests have become detached.



*Two Nægele Pelves.*

PLATE 4.



PELVIS B.—Fig. 2.

Note the absence of the tuberosity of the ilium. The prominent end of the crest is not the posterior superior spine; its site is just above the level of the second sacral foramina. The straightness and upward displacement of the right ilium are well shown.





# WIDAL'S REACTION.

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A CRITICAL EXAMINATION OF 326 CASES IN WHICH THE  
REACTION HAS BEEN TRIED.

By W. C. C. PAKES.

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IN 1897<sup>1</sup> I published an account of the results which I had obtained with the typhoid serum reaction. Since that paper was published there has been a more or less systematic examination of the cases of enteric fever admitted into Guy's Hospital, and of many cases which, during some period of the disease, might have been enteric fever.

Three hundred and seventy-five cases have been examined, many of them on more than one occasion, and the present paper deals with 326 of these, the remaining 51 I have been unable to discover in the Reports.

*The collection of the serum.*—The serum is collected by the house physicians, clinical clerks, or ward clerks, in the special pipettes which I have previously described. They are larger than those in general use, and are so made that when the blood is collected it can be allowed to coagulate in the middle, so that the serum can separate, and run down into the finer part free from corpuscles.

*The dilution of the serum.*—In order to dilute the serum, small test-tubes and small graduated pipettes are used. Ten cubic millimetres of serum are pipetted into a small test-tube, and are mixed with 90 cubic millimetres of broth, which is measured in a

<sup>1</sup> *Lancet*, May 29th, 1897.

second pipette. This gives a fluid containing 10 per cent. of serum; 10 cubic millimetres of this 10 per cent. serum are pipetted into a second test-tube, and are mixed with 90 cubic millimetres of broth. This gives a fluid containing 1 per cent. of serum.

Sterile broth is used instead of water to prevent the laking of the red discs. It was shown soon after the reaction was discovered that the red discs contained more of the agglutinins than the clear serum, and if sometimes laked blood is present, and at other times no blood, the observations cannot be said to be made under identical conditions, and the value of the reaction as a diagnostic test will be impaired.

*The culture of the B. typhi abdominalis.*—The whole of the observations have been made with one of two strains, which we call "A" and "3." The first, "A," is the same strain which I used in my first series in 1897. The second is one which was isolated from the spleen of a patient dead of enteric fever. Observations have been made with other strains, but the results in the tables are entirely with these two strains. The bacilli are inoculated on agar every fortnight, and grown at 20° C. As cultures are required for the reaction, broth tubes are inoculated from the most recent agar culture about eighteen hours before they are required, the broth tubes being incubated at 37° C.

*Mixing the serum with the broth culture.*—Three microscopic examinations have been made latterly, the serum in the mixtures being in the proportion of 50 per cent., 5 per cent., and 0·5 per cent. In order to do this, a platinum loopful of the undiluted serum is mixed upon a clean coverslip with the same loop full of the broth culture, thus making the 50 per cent. dilution. One loopful of the 10 per cent. serum and of the 1 per cent. serum mixed with a loopful of the broth culture form a 5 per cent. and 0·5 per cent. dilution respectively. Immediately after mixing, the coverslips are inverted upon a hanging drop slide, and the time of mixing noted.

*The reaction, and reaction time.*—When judging of a reaction it is necessary that practically the whole of the bacilli shall be grouped together, leaving only isolated single bacilli free in the

field. If there are a few small clumps, but the majority of the bacilli are either motile or free in the field, I have called this a half reaction, " $\frac{1}{2} +$ ." As regards the time, I have always been very rigid, and have continued to adopt the time I suggested in 1897, viz., half an hour. If there is not a complete reaction within half an hour, even if it is complete within one or two hours, I have returned the result as " $\frac{1}{2} +$ ," that is, "no reaction."

*The diagnosis of the disease.*—In those cases where a post-mortem examination has been performed, the diagnosis has been made from the result of the autopsy. When the patient has recovered, the diagnosis has been arrived at from the consideration of the whole of the symptoms and course of the disease after its termination. Provisional diagnoses during the course of the disease have not been accepted unless the physician has been satisfied, after the termination of the case, that it was really enterica or was really not enterica.

*Explanation of the tables.*—Under the heading *Reaction* there are three divisions, viz., 50 per cent., 5 per cent., and 0.5 per cent. It will be noticed that these are not all filled in. The reasons for this fact are two; first, the 0.5 per cent. dilution was not systematically performed until the middle of 1898; and secondly, sufficient serum was not always obtained to enable all the dilutions to be made.

Each case has only one reaction in the tables, although many of them were tried two, three, or even four times. In most cases the last of the reactions has been tabulated, especially when this has differed from previous ones. For instance, as will be seen hereafter, a case of enteric was examined upon the fourth, ninth, and thirteenth days. On the first occasion the reaction was  $+: \circ: \circ$ ; on the second,  $+: +: \circ$ ; and on the third,  $++: ++: +$ . The third of these appears in the tables.

The headings *Spleen felt* and *Typhoid spots* explain themselves. The only points which it is necessary to explain, are first, that " $+$ " means that they were felt or seen during some period of the disease, and not necessarily at the time when the serum was examined. In many of the cases I have been unable to find any observation concerning these points in the reports;

this is indicated by the sign "—"; when it is stated "the spleen could not be felt," or "no typhoid spots were seen," these facts have been indicated by the sign "O."

#### ANALYSIS OF THE TABLES.

The cases have been tabulated in five divisions:—

- I. Cases in which an autopsy has been held.
- II. Cases of enteric fever, which gave a reaction.
- III. Cases which were not enteric fever, and which gave no reaction.
- IV. Cases of enteric fever which gave no reaction.
- V. Cases which, not being enteric fever, gave a reaction.
- VI. Cases which remained undiagnosed.

#### DIVISION I.—Cases where a post-mortem has been held.

No.	Reaction.			Spleen felt.	Typhoid spots.	Result of post-mortem examination.
	50 per cent.	5 per cent	0.5 per cent.			
1	+	O	—	—	—	Acute suppurative pancreatitis.
2	++	++	—	—	O	Enteric. Ulceration of small intestine—General supp. peritonitis.
3	O	O	—	—	—	General tuberculosis.
4	++	+	—	+	+	Enteric. Ulceration of small and large intestine—perforation.
5	++	+	—	—	+	Enteric. Ulceration of small and large intestine.
6	$\frac{1}{2}$ +	O	—	+	—	Infective endocarditis. Intestines normal.
7	++	++	—	+	—	Enteric. No intestinal lesions. B.t.a. isolated from enlarged mesenteric glands.
8	++	++	+	+	?+	Enteric. Ulceration of intestine—large spleen.
9	++	++	+	+	+	Enteric. Comparatively small area of intestinal ulceration. Much ante-mortem thrombosis of lungs.
10	++	+	+	—	O	Enteric. Ulceration of intestine.
11	O	O	O	—	—	Pneumonia. Intestines distended but not ulcerated.
12	O	O	—	—	—	Double lobar pneumonia.
13	++	+	$\frac{1}{2}$ +	O	O	Enteric. Six round ulcers in intestine, one perforated.
14	++	++	+	—	+	Enteric. B.t.a. found in spleen.
15	$\frac{1}{2}$ +	O	O	—	—	General pyæmia. Abscesses in right lung and left kidney.
16	+	+	+	?	+	Enteric. Ulceration of intestines.
17	+	O	O	—	—	Lobar pneumonia and emphysema.

## DIVISION I.—continued.

No.	Reaction.			Spleen felt.	Typhoid spots.	Result of post-mortem examination.
	50 per cent.	5 per cent.	0·5 per cent.			
18	++	++	+	+	+	No trace of ulceration past or present. Spleen soft, but little—if any—enlarged.
19	$\frac{1}{2}$ +	○	○	—	—	Pericarditis and acute endocarditis.
20	+	○	○	—	—	Tuberculous peritonitis.
21	○	○	○	—	—	Tuberculous peritonitis.
22	++	+	○	+	—	Enteric. Ulceration of intestines.
23	○	○	○	—	—	Enteric. Ulceration of intestines—double lobar pneumonia.
24	—	$\frac{1}{2}$ +	○	—	—	Enteric. Ulceration of intestines—pneumonia.
25	$\frac{1}{2}$ +	○	○	—	—	Tuberculous peritonitis.
26	○	○	○	—	+	Enteric. Ulceration of intestines—pneumonia.
27	+	$\frac{1}{2}$ +	○	○	○	Actinomycosis of liver with localised peritonitis.
28	+	+	○	—	+	Typhus. Congested mucous membrane of intestine with small petechiæ, but no inflammation of Peyer's patches or solitary follicles.
29	—	$\frac{1}{2}$ +	○	—	—	Meningitis.
30	+	○	○	—	—	Malignant growth of lung.

It will be seen from the above table that of the thirty cases twelve had post-mortem indications of enteric fever, and all yielded positive reactions in life. Fourteen had post-mortem indications of some disease not enterica, and none of these gave any serum reaction in life. In four cases the post-mortem appearances did not agree with the serum reaction. Two of these, cases 23 and 24, can be eliminated, because the examination was made in each case on the seventh day of the disease, and not at a later period. The well-known fact that undoubted cases of enterica do not show a reaction during the earlier stages is well seen in this series and will be alluded to later. The two anomalous cases remaining are Nos. 26 and 28.

CASE 26.—Henry C., æt. 24, had what appeared to be an ordinary attack of enteric fever, with spots and a typical temperature. After about a fortnight he developed pneumonia, of which he really died. The serum was examined upon two occasions corresponding to the eighth and sixteenth days of the disease, but gave no reaction.

CASE 28.—Richard W., æt. 41, was admitted to the hospital suffering from what was thought to be enteric fever, but which was subsequently diagnosed

to be typhus fever. In the report it is stated that at first there was a red lenticular rash, fading on pressure, over the abdomen, back, and the posterior surface of upper arms; this was succeeded by a subcuticular brownish-red mottling. The serum reaction was given on the ninth day of the disease, and the patient died of hyperpyrexia on the tenth day of the disease. No previous history could be obtained from the patient or his relatives, the former being too ill, and the latter being at the time in several of the Metropolitan Asylums Board's hospitals suffering from typhus fever.

CASE 26 is, I think, an illustration of the theory to which I have previously assented, that certain cases which terminate fatally never give a reaction, because the body never produces the agglutinins in sufficient quantity to become evident. The reaction, of which the presence of agglutinins is evidence, is one of immunity; if, therefore, the patient never attempts to acquire an immunity, that is, that the tissues never attempt to resist the infection, Widal's reaction will never be obtained. These cases will necessarily be fatal.

CASE 28 raises some interesting points. In the first place, the patient may have previously had enteric fever. On the other hand, the history of the case shows that the patient was at first thought to be suffering from enteric, chiefly on account of the typical typhoid spots. The appearance of the petechial rash was followed by a change in diagnosis to typhus. If the disease was typhus, it must be allowed that the rash of this disease may be indistinguishable from that of enteric fever. The post-mortem appearances and the fact that other members of the family were suffering from typhus fever point to the fact that the patient had typhus fever. It seems to me to be possible that the patient had enteric fever, and that during the incubation of this disease he contracted typhus, the typhus being superimposed upon the enteric. It is well known that diphtheria and enteric, scarlet fever and enteric, measles and diphtheria and other infectious diseases may co-exist.

Two more interesting cases appear in this table, Nos. 7 and 18. No. 7 has been published by Dr. Bryant<sup>2</sup> as a case of enteric fever without intestinal lesions. No. 18 is another case of the same kind.

<sup>2</sup> Brit. Med. Journal, April 1, 1899.

CASE 18.—Thomas H., æt. 26, had what appeared to be a typical but severe attack of enteric fever. The temperature chart was typical and the stools were "typhoid like;" in addition to this the spleen was felt during life, and the patient had numerous typhoid spots. At the autopsy no intestinal lesions were found, the spleen was soft and slightly enlarged. Begging the question of the paper for a moment, very strong presumptive evidence is furnished by the fact that the serum of the patient reacted in a strength of 1 in 200.

DIVISION II.—Cases of Enteric Fever giving a positive Widal Reaction.

No.	Reaction.			Spleen felt.	Typhoid spots.	Diagnosis.
	50 per cent.	5 per cent.	0.5 per cent.			
31	+	+	—	○	+	Enteric fever.
32	++	+	—	+	—	"
33	++	++	—	—	—	"
34	+	+	—	+	—	"
35	++	++	—	+	+	"
36	+	+	+	+	+	"
37	+	+	—	—	—	"
38	+	+	—	+	+	"
39	++	++	—	+	+	"
40	+	+	—	+	+	"
41	+	+	—	—	+	"
42	++	++	—	+	+	"
43	+	+	—	+	+	"
44	++	++	+	—	—	"
45	—	+	—	—	+	"
46	+	+	—	—	+	"
47	++	++	—	+	—	"
48	++	+	—	+	—	"
49	—	++	—	—	—	"
50	++	+	—	+	—	"
51	++	+	—	+	+	"
52	++	+	—	+	+	"
53	++	+	—	+	+	"
54	++	++	—	+	+	"
55	++	+	—	—	+	"
56	+	+	—	—	+	"
57	++	+	—	—	+	"
58	++	+	—	—	+	"
59	++	+	—	+	—	"
60	++	+	—	+	—	"
61	+	+	—	+	—	"
62	++	+	—	+	+	bronchitis.
63	—	+	—	+	+	"
64	++	+	—	+	+	"
65	++	+	—	—	—	"
66	++	++	—	+	—	"
67	++	+	—	—	+	"
68	++	++	—	+	—	"
69	++	+	—	—	+	"
70	++	+	—	+	+	"
71	++	+	—	—	—	"

## DIVISION II.—continued.

No.	Reaction.			Spleen felt.	Typhoid spots.	Diagnosis.
	50 per cent.	5 per cent.	0.5 per cent.			
72	++	+	—	—	—	Enteric fever.
73	++	++	—	—	—	"
74	++	+	—	—	+	"
75	+	+	—	—	+	"
76	+	+	—	—	—	"
77	++	++	+	—	+	"
78	++	+	—	+	—	"
79	++	++	—	—	+	"
80	+	+	—	+	+	"
81	++	+	—	—	—	"
82	+	+	—	+	+	"
83	++	++	+	+	+	"
84	++	+	+	—	+	"
85	++	++	++	+	+	"
86	++	++	—	—	+	"
87	+	+	○	—	+	"
88	++	++	+	+	+	bronchopneumonia.
89	++	+	+	+	+	"
90	++	+	+	—	+	"
91	+	+	○	—	+	"
92	++	+	+	+	—	"
93	—	+	+	+	+	"
94	++	++	+	—	+	"
95	++	+	$\frac{1}{2}$ +	+	+	"
96	++	+	+	+	—	"
97	++	+	—	—	—	"
98	++	+	+	+	+	"
99	++	+	+	+	+	"
100	+	+	○	+	+	"
101	++	++	+	+	+	relapse.
102	+	+	○	—	+	"
103	++	++	+	+	+	"
104	++	++	+	—	+	with pneumonia.
105	++	+	$\frac{1}{2}$ +	—	+	"
106	++	+	+	+	+	"
107	++	+	—	—	—	"
108	++	+	—	+	—	"
109	++	+	$\frac{1}{2}$ +	+	+	"
110	++	+	+	—	+	hyperpyrexia.
111	++	++	+	—	+	subsequent to acute rheumatism.
112	++	+	○	+	+	"
113	++	++	+	—	+	relapse.
114	++	++	+	+	+	"
115	++	++	+	+	+	"
116	+	+	+	+	—	"
117	++	++	$\frac{1}{2}$ +	—	+	"
118	++	+	+	—	+	"
119	++	+	+	—	+	"
120	++	+	○	—	+	hyperpyrexia.
121	+	+	○	—	?	thrombosis of left femoral vein.



## DIVISION II.—continued.

No.	Reaction.			Spleen felt.	Typhoid spots.	Diagnosis.
	50 per cent.	5 per cent.	0.5 per cent.			
122	++	++	—	—	+	Enteric fever.
123	++	++	++	—	+	"
124	+	+	+	—	+	"
125	++	+	+	—	+	"
126	+	+	○	+	—	"
127	++	+	+	+	—	"
128	++	+	+	—	+	" relapse.
129	+	+	○	—	+	"
130	++	++	+	+	+	"
131	++	+	+	+	+	"
132	++	++	++	—	—	"
133	++	++	+	+	+	"
134	++	++	+	—	+	"
135	—	++	+	—	+	"
136	+	+	+	—	—	"
137	+	+	+	—	—	"
138	+	+	$\frac{1}{2}$ +	—	—	"
139	++	+	+	—	—	"
140	+	+	○	+	?	"
141	++	+	—	—	—	" hæmorrhage.
142	++	++	+	+	+	"
143	+	+	$\frac{1}{2}$ +	+	+	"
144	+	+	+	—	—	" hæmorrhage.
145	—	+	○	+	+	"
146	++	+	+	?	+	"
147	++	+	+	—	+	"
148	++	+	○	—	+	"
149	++	++	$\frac{1}{2}$ +	—	+	" hæmorrhage.
150	+	+	○	—	+	"
151	+	+	$\frac{1}{2}$ +	—	+	" hæmorrhage.
152	+	+	○	+	—	" bronchitis.
153	++	+	+	—	+	"
154	+	+	$\frac{1}{2}$ +	—	—	"
155	++	$\frac{1}{2}$ +	$\frac{1}{2}$ +	+	—	"
156	—	+	+	+	—	" relapse.
157	—	+	+	+	+	"
158	—	+	+	—	+	"
159	—	+	○	—	—	"
160	+	+	○	—	—	" hæmorrhage—appen- dicular abscess.
161	+	+	+	—	+	" relapse.
162	+	+	+	—	—	"
163	+	+	$\frac{1}{2}$ +	+	—	"
164	—	+	+	+	+	"
165	++	+	$\frac{1}{2}$ +	—	—	" relapse.
166	+	+	+	—	+	"
167	—	+	+	+	+	"
168	++	+	+	—	+	"
169	+	+	$\frac{1}{2}$ +	—	+	"
170	—	+	$\frac{1}{2}$ +	+	+	"
171	+	+	$\frac{1}{2}$ +	+	+	"
172	+	+	+	+	+	"

## DIVISION II.—continued.

No.	Reaction.			Spleen felt.	Typhoid spots.	Diagnosis.
	50 per cent.	5 per cent.	0·5 per cent.			
173	+	+	+	+	+	Enteric fever.
174	+	+	○	—	—	"
175	+	+	$\frac{1}{2}$ +	—	—	"
176	+	+	+	—	?	"
177	—	+	—	—	+	"
178	+	+	$\frac{1}{2}$ +	○	+	"
179	—	+	○	○	+	"
180	—	+	+	○	+	"
181	+	+	$\frac{1}{2}$ +	—	—	"
182	+	+	$\frac{1}{2}$ +	—	?	"
183	+	+	$\frac{1}{2}$ +	○	—	"
184	++	+	+	—	—	delirium.
185	+	+	○	+	+	"
186	++	++	+	+	+	"
187	+	+	+	—	—	"
188	+	+	+	+	+	"
189	++	+	—	+	+	"
190	+	+	$\frac{1}{2}$ +	○	+	"
191	+	+	+	+	+	"
192	++	+	$\frac{1}{2}$ +	—	+	"
193	+	+	+	—	—	"
194	+	+	+	+	+	"
195	++	+	+	+	+	"
196	+	+	+	○	+	"
197	—	+	○	—	+	"
198	+	+	+	○	○	"
199	+	+	$\frac{1}{2}$ +	○	—	bronchopneumonia.
200	—	+	$\frac{1}{2}$ +	—	+	"
201	+	+	—	+	—	"

The 171 cases included in this division require little comment. The reaction given by the serum was indisputable. Of the total 326 cases the 0·5 per cent. dilution has been tried on 218 occasions, 124 in cases of enteric and 94 in cases not of enteric. Of the 124 examinations in cases of enteric, 75 yielded a positive result and 49 a negative result. Of the 94 examinations in cases not of enteric, no case gave a positive result. From these figures I think it is fair to conclude that *if any serum gives a reaction in a strength of 0·5 per cent. the patient yielding the serum has, or has suffered from enteric fever.*

## DIVISION III.—Cases not Enteric giving no Widal Reaction.

No.	Reaction.			Spleen felt.	Typhoid spots.	Diagnosis.
	50 per cent.	5 per cent.	0·5 per cent.			
202	○	○	—	—	—	Colitis.
203	+	○	—	+	—	Bronchitis.
204	+	○	—	—	—	Locomotor ataxia. Influenza.
205	○	○	—	—	—	Ulcerative colitis.
206	○	○	—	+	—	Tuberculous peritonitis.
207	○	○	—	+	—	Pyrexia. Previously had malaria.
208	○	○	—	—	—	Apical pneumonia.
209	+	○	—	—	—	Chronic alcoholism.
210	+	○	—	—	—	Hyperpyrexia. ? Cause.
211	○	○	—	—	—	Influenza.
212	○	○	—	—	—	Pneumonia.
213	○	○	—	+	—	Pyrexia. ? Influenza.
214	○	○	—	+	—	Ascites with pyrexia.
215	+	○	—	—	—	Tumour in right iliac fossa.
216	+	○	—	—	—	No diagnosis, but not enterica.
217	○	○	—	—	—	Right lobar pneumonia.
218	○	○	—	—	—	Pyrexia.
219	+	○	—	—	—	Lobar pneumonia.
220	$\frac{1}{2}$ +	○	—	—	—	Typhus fever.
221	○	○	—	—	—	Influenza.
222	+	○	—	—	—	Emphysema. Acute bronchitis.
223	+	○	○	—	—	Ptomaine poisoning.
224	○	○	—	—	—	Lobar pneumonia.
225	+	○	○	—	—	Tonsillitis.
226	○	○	○	+	—	Typhus fever.
227	○	○	○	+	○	Typhus fever.
228	+	○	—	+	—	Influenza.
229	$\frac{1}{2}$ +	○	—	—	—	Pleurisy with effusion.
230	$\frac{1}{2}$ +	○	—	+	—	Enlarged liver. ? Cirrhosis.
231	+	○	—	—	—	Lumbar pain. Pyrexia.
232	○	○	○	—	—	Lobar pneumonia.
233	+	○	—	—	—	Pyrexia. ? Cause.
234	○	○	—	—	—	Pyrexia. ? Cause.
235	○	○	—	—	—	Lobar pneumonia.
236	○	○	○	—	—	Acute gastritis.
237	$\frac{1}{2}$ +	○	—	—	—	? Carcinoma ventriculi.
238	$\frac{1}{2}$ +	○	—	—	—	Acute pulmonary tuberculosis.
239	$\frac{1}{2}$ +	○	○	—	—	Pyrexia.
240	○	○	○	—	—	Lobar pneumonia.
241	+	○	—	—	—	Pericarditis.
242	$\frac{1}{2}$ +	○	○	—	—	Pleuritic effusion.
243	○	○	○	—	—	Pyrexia.
244	+	○	—	—	—	No diagnosis not enterica.
245	$\frac{1}{2}$ +	○	○	—	—	Right apical pneumonia.
246	$\frac{1}{2}$ +	○	—	—	—	Pleuritic effusion.
247	$\frac{1}{2}$ +	○	○	—	—	Acute hydronephrosis.
248	$\frac{1}{2}$ +	○	○	—	—	? Cirrhosis of liver.
249	○	○	○	—	—	Intussusception.
250	$\frac{1}{2}$ +	○	○	+	—	Headache. No temperature.
251	○	○	○	—	—	Pyrexia.
252	○	○	○	—	—	Lobar pneumonia.
253	+	○	○	—	—	Thrombosis of femoral vein.

## DIVISION III.—continued.

No.	Reaction.			Spleen felt.	Typhoid spots.	Diagnosis.
	50 per cent.	5 per cent.	0.5 per cent.			
254	$\frac{1}{2}+$	○	○	—	—	Pleuritic effusion.
255	$\frac{1}{2}+$	○	○	—	—	Gonorrhœal arthritis. Malignant endocarditis.
256	+	○	○	—	—	Acute gastritis.
257	—	○	○	—	—	Bronchopneumonia.
258	+	○	○	—	—	Fits.
259	+	○	○	—	—	Influenza.
260	$\frac{1}{2}+$	○	○	—	—	Cerebral tumour. Purulent meningitis.
261	+	○	○	+	○	Pertussis—bronchitis.
262	○	○	○	+	—	Anæmia. High intermittent temperature.
263	○	○	○	—	—	Chronic tubal nephritis.
264	+	○	○	—	—	Staphylococcal abscess in right loin.
265	+	○	○	—	—	Pleural effusion.
266	+	$\frac{1}{2}+$	○	—	—	Pyrexia.
267	○	○	○	—	—	Epidemic enteritis.
268	○	○	○	+	—	? Septicæmia.
269	+	○	○	—	—	Alcoholic peripheral neuritis.
270	$\frac{1}{2}+$	○	○	—	—	Pyrexia. ? Cause.
271	○	○	○	—	—	Lobar pneumonia.
272	+	○	○	—	—	No diagnosis. Not enterica.
273	+	○	○	○	○	Gonorrhœal arthritis.
274	—	$\frac{1}{2}+$	○	—	—	Lobar pneumonia. Pleurisy.
275	+	○	○	—	—	Appendicitis.
276	+	○	○	—	—	Basal meningitis.
277	—	○	○	+	—	Malaria—Blackwater.
278	+	$\frac{1}{2}+$	○	—	—	Pleuritic effusion.
279	+	○	○	—	—	Jacksonian epilepsy.
280	+	$\frac{1}{2}+$	○	—	—	Pneumonia. Herpes labialis.
281	+	$\frac{1}{2}+$	○	—	—	Apical pneumonia. Herpes.
282	+	$\frac{1}{2}+$	○	—	—	Cirrhosis. Lobar pneumonia.
283	○	○	○	—	—	Acute rheumatism.
284	○	○	○	—	—	Appendicitis.
285	+	○	○	—	—	Chlorosis.
286	+	○	○	—	—	Phthisis—diarrhœa.
287	+	○	○	—	—	Acute bronchitis.
288	+	○	○	—	—	Diarrhœa and vomiting—pleurisy.
289	—	○	○	—	—	Appendicitis.
290	—	$\frac{1}{2}+$	○	—	—	? Rheumatism. Convalescent when admitted.
291	○	○	○	—	—	Typhus fever.
292	+	$\frac{1}{2}+$	○	—	—	Emphysema.

This division calls for but few remarks. The diseases from which the patients have suffered have been most varied, but in no single instance has the serum given the reaction. Although the fact is not shown in the tables, many of these cases have been

examined on two or more occasions with the same results. It will be seen that of these cases thirty-nine gave a reaction when the serum was in a strength of 50 per cent., and eight gave a partial reaction when the serum was in a strength of 5 per cent. These facts are further confirmation of the so often reiterated statement that when this test is used for the purposes of diagnosis the serum must be sufficiently diluted or the result of the examination may be valueless. Of the eight partial reactions with 5 per cent. serum, three at least gave a complete reaction after from six to twenty-four hours; this bears out what I said in 1897, that a strict time limit should be enforced, and I have had no reason to alter the time which I suggested, namely, half an hour.

DIVISION IV.—Cases of Enteric Fever which gave no  
Widal Reaction.

No.	Reaction.			Spleen felt.	Typhoid Spots.	Diagnosis.	Day of Disease.
	50 per cent.	5 per cent.	0.5 per cent.				
293	+	○	—	—	+	Enteric fever ...	9th or 10th <sup>*</sup>
294	+	○	—	+	+	" ...	11th
295	+	○	—	—	+	" ...	16th
296	$\frac{1}{2}$ +	○	—	+	○	" ...	13th
297	+	$\frac{1}{2}$ +	○	—	+	" ...	7th
298	+	○	○	○	○	" ...	19th
299	+	○	○	+	+	" Very mild case.	4th, 10th, 18th
300	○	○	—	+	+	" ...	25th or 26th
301	$\frac{1}{2}$ +	○	○	—	+	" ...	11th & 19th
302	$\frac{1}{2}$ +	○	○	+	+	" ...	7th, 15th, 29th
303	+	$\frac{1}{2}$ +	○	—	1 spot	" ...	11th
304	+	$\frac{1}{2}$ +	○	○	○	" ...	18th
305	○	○	○	+	+	" ...	4th
306	○	○	○	+	+	" ...	10th & 23rd
307	○	○	○	—	—	" ...	4th
308	—	○	○	—	?	" Very mild case.	22nd
309	○	○	○	+	+	" ...	6th or 7th

Of the seventeen cases included in the above list, a few, five or six in number, were diagnosed as most probably enteric, after a careful consideration of all the facts. I have included them in this division rather than in division VI., so that the error will be overstated rather than understated.

It is well known that many cases of undoubted enteric fever do not give any reaction before the tenth or eleventh day of the disease, and it is my custom to inquire what the day of the disease is, in order that subsequent examinations may be made. I shall allude to some of these cases later. For the present, I merely mention the fact in justification of the statement that the examinations made on or before the eleventh day of the disease are—if a negative result is obtained—of no value. We must therefore exclude cases 293, 294, 297, 303, 305, 307 and 309, which were all typical cases of enteric, but whose sera were not examined after the eleventh day of the disease.

CASE 295 was a typical case of enteric, but no reaction was obtained on the sixteenth day of the disease.

CASE 296 was undoubtedly enteric fever, but no reaction was obtained on the thirteenth day of the disease.

CASE 298.—Chas. J., *æt.* 37, is said to have had enteric fever five years previously. He had been ill for ten days before admission, with cough, headache and dyspnoea. For the first four days in the hospital the temperature varied between  $99^{\circ}$  and  $103^{\circ}$ , after this the remissions were less marked, and seventeen days after admission to the hospital the temperature was normal. The spleen was never felt, nor were any typhoid spots seen. On three or four occasions loose light-coloured stools were passed. On the nineteenth day of the disease the serum gave no reaction.

CASE 299.—Fred S., *æt.* 15. The spleen was felt, and numerous typhoid spots were seen. The highest temperature was  $102^{\circ}$  on the ninth and tenth days of the disease; on the eighth, eleventh, twelfth and thirteenth days the temperature was  $101^{\circ}$ , and on the fifteenth day  $100^{\circ}$ . The patient was never seriously ill, and the case was a very mild one. No reaction was obtained on the fourth, tenth and eighteenth days of the disease.

CASE 300.—Ray A., *æt.* 3. This was an uncomplicated case of enteric fever, with a temperature of  $100^{\circ}$  F. for ten or probably twelve days, from October 22nd or 24th to November 4th. Several typical spots were observed, and the spleen was enlarged. The examination was made on the twenty-fifth or twenty-sixth day of the disease, and there was no reaction.

CASE 301.—Francis F., *æt.* 4. Had a temperature of over  $103^{\circ}$  for ten days, and was frequently sponged during this time on account of hyperpyrexia. On the fourteenth day of disease the temperature became normal, and remained so until the twenty-first day. On this day the temperature suddenly rose to  $104^{\circ}$ , and during the next four days varied between  $104^{\circ}$  and  $99.4$ ; on the thirty-first day it suddenly dropped to normal, rising again on the following day to  $102^{\circ}$ , and for the next seven days the temperature gradually fell to normal, and from this day onwards the patient made an uninterrupted recovery. A few doubtful spots were seen, but the spleen was not felt. On the eleventh day of disease there was only a very partial reaction with the 50 per cent. serum, and no reaction with the 5 per cent. On the nineteenth day the 50 per cent. serum gave a complete reaction, and

the 5 per cent. a partial reaction. Unfortunately, no subsequent examination was made.

CASE 302.—Richard N., æt. 2. This patient had a typical temperature for twelve days. After convalescence had set in there was a rise in temperature for four days. This was looked upon as the relapse. The spleen was felt and several typhoid spots were seen. No reaction was obtained on the seventh, fifteenth and twenty-ninth days.

CASE 304.—Ellen P., æt. 25. This case was one which was diagnosed rather by exclusion than by any definite symptoms. The spleen was not felt, no spots appeared, and the temperature was of a distinctly "septic" type. The serum was examined on the tenth and eighteenth days, but gave no reaction.

CASE 306.—Eliz. F., æt. 4. The spleen was felt, and the patient had several typhoid spots. The temperature was markedly intermittent. The serum was examined on the tenth and twenty-third days, and gave no reaction.

CASE 308.—George O., æt. 31. The patient was a worker in drains, and had sixteen days' pyrexia, 103° being the highest temperature recorded. No remark is made about the spleen, and doubtful typhoid spots were seen.

#### DIVISION V.—Cases which were not Enteric, but which gave Widal's Reaction.

No.	Reaction.			Spleen felt.	Typhoid spots.	Diagnosis.	Day of Disease.
	50 per cent.	5 per cent.	0·5 per cent.				
310	+	+	○	○	?	Acute rheumatism.	9th.
311	+	+	○	○	○	Lobar pneumonia.	
312	+	+	○	—	—	Lobar pneumonia.	
313	++	+	○	—	—	Broncho-pneumonia.	
314	+	+	○	—	—	Appendicular abscess	

Of the five cases included in this division one, namely case 311, can be withdrawn, since the patient had enteric fever a year before admission into the hospital. In this division 313 has been included, instead of being included in the next division, because it is probable that the disease was not one of enteric.

CASE 310.—Fanny P., æt. 41. Patient had had scarlet fever and diphtheria, but there was no history of previous enteric. It is true that the case was diagnosed as acute rheumatism, and that it got better under salicylates; but the temperature chart was exceedingly suspicious of enteric, and before the final diagnosis of acute rheumatism was made, it was stated in the report that there were a few doubtful typhoid spots. The serum was tested on the ninth day of the disease, and gave a reaction.

CASE 312.—Mary B., æt. 40. This was a somewhat complicated case, and among other things was certainly one of lobar pneumonia. There was, however, an enlarged spleen and considerable diarrhoea. Although there is a

doubt as to the absence of enteric, the evidence on the whole points to the fact that it was not a case of enteric. The patient's history is somewhat interesting, because she had been in Stockwell twenty-two years previously, and ten months previous to admission into Guy's had suffered from what she described as severe diarrhoea. It seems almost impossible that the reaction should have lasted for twenty-two years, and if we assume that the disease ten months previously was enteric, we have to allow that it is one of the rare cases of typhoid occurring twice in twenty-four years.

CASE 313.—George W., *æt.* 4. Nine months previous to admission into the hospital patient had measles, and the mother said that it took a long time to recover, and had not been really well since: there is, however, no evidence beyond this either in favour of or against the patient having had enteric fever.

CASE 314.—George T., *æt.* 18. This was a case of appendicular abscess, complicated, however, by hæmorrhage from the bowels. Although in the early stage of the disease enteric was considered, the probabilities are that the whole disease arose from the appendix and was not enteric fever.

DIVISION VI.—Cases in which Enteric Fever could neither  
be asserted nor excluded.

No.	Reaction.			Spleen felt.	Typhoid spots.	Diagnosis.	Day of Disease.
	50 per cent.	5 per cent.	0.5 per cent.				
315	++	+	—	—	?	? Enteric ... ..	12th.
316	+	○	○	—	?		
317	○	○	○	○	○	Febricula ... ..	13th.
318	+	+	○	—	—		
319	+	+	○	—	?	Bronchitis. ?Tuber- cular pyrexia.	
320	+	+	½+	—	—		
321	+	○	—	—	—		10th.
322	+	○	○	○	○		16th.
323	++	+	—	○	—		3rd month.
324	+	○	○	—	?		10th & 18th days
325	○	○	○	○	+		
326	○	○	○	—	—		

This division includes those cases which were undiagnosed by the physician; as will be seen later, these cases are subtracted from the total number before the percentage error is calculated.

CASE 320.—The history of this case is incomplete, but a consideration of the whole facts appears to allow the deduction that the patients had enteric fever and recovered. During the convalescent stage she aborted and died of pyæmia contracted subsequent to the abortion.

In three of the cases, Nos. 315, 318, and 319, the disease was eventually diagnosed as enteric, but simply on account of the reaction which their sera gave.



There are a few points of interest in the series which may be very briefly mentioned.

In the first place there seems to be no fixed period for the appearance of the reaction in cases of undoubted enteric fever. In Division II. the earliest appearance of the reaction in the 5 per cent. dilution was on the fifth day of the disease, and the earliest appearance in the 0·5 per cent. dilution was on the eighth day. The latest appearance of the reaction in the 5 per cent. dilution was on the eighteenth or nineteenth day. It is very rarely that one finds the reaction coming on as late as this, but the fact that out of the 171 cases it has been found in two cases as late as this justifies me, I think, in deducting several of the cases in Division IV. before calculating the minimum error. In many of the cases in Division II. no reaction was ever obtained in the 0·5 per cent. dilution even as late as the second month, and even though the patient had one or more relapse.

The present series also shows what has been shown before, that the intensity of the reaction varies from time to time during the progress of the disease. On several occasions in the 0·5 per cent. dilution there has been a reaction on, say, the fourteenth day, an incomplete reaction on the twentieth day, and again a complete reaction on the thirtieth day. One case gave (as the strongest reaction) a reaction in 1 per cent. dilution on the twelfth day; on the eighteenth day there was a reaction in 0·25 per cent., on the twenty-fourth day only in 0·5 per cent., and on the thirty-first day in 0·125 per cent.

The different strains of the *B. typhi abdominalis* have a distinct influence. On several occasions I have found that "3" gave a reaction in the 0·5 per cent. dilution when "A" "B" "X" and "Z" gave no reaction in a greater dilution than 5 per cent., or 2·5 per cent. On five occasions "A" and "3" gave a reaction in 5 per cent. when neither "B" "C" nor "Z" gave a reaction. There is therefore an undoubted fallacy, however small it may be, in performing the test with only one strain of the bacillus, and I think that it is highly probable that some of the cases in Division IV. would have been placed in Division II. if I had been able to try several strains.

*The error found.*—In order to calculate the error of Widal's reaction from the present series, it is first necessary to make certain deductions, namely, the eleven doubtful cases forming Division VI.; nine of the cases in Division IV., since these were examined with a negative result before the eleventh day of the disease; and one case in Division V. as the patient had previously suffered from enteric fever. The figures to be used, therefore, in calculating the error are as follows:—

Division	I.	Agreeing (positive).	Agreeing (negative).	Disagreeing.	Total.
	I.	12	14	2	28
	II.	171	0	0	171
	III.	0	91	0	91
	IV.	0	0	10	10
	V.	0	0	4	4
	VI.	0	0	0	0
		<hr/>	<hr/>	<hr/>	<hr/>
		183	105	16	304

The error obtained from these figures, — 16 out of 304, is 5·26 per cent. The maximum deviation from this figure—that is, the error possible on account of the comparatively small number of cases—is 3·6 per cent., in other words, the error lies between 1·6 per cent. and 8·8 per cent. This represents the *maximum error* since several cases are still included which were probably enteric fever, but which were not proved conclusively to have been enteric fever.

In order to obtain the *minimum error* these cases must be deducted: they are seven in number, namely, 298, 304, 308, 310, 312, 313 and 314. This leaves us with 297 cases of which 9 gave a reaction not in accord with the diagnosis. The error calculated from these figures is 3·03 per cent. and lies between 0·2 per cent. and 5·8 per cent.

# CHRONIC MERCURIAL POISONING, WITH SPECIAL REFERENCE TO THE DANGER IN HATTERS' FURRIERS' MANUFACTORIES.

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(THESIS FOR M.D. CANTAB.)

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MERCURIAL poisoning occurs in two forms, the acute, and the chronic. Acute mercurial poisoning is characterised by severe gastro-intestinal irritation, with collapse.

Chronic mercurial poisoning may take one of two forms, or the two forms may be combined. One is characterised by salivation and sore gums, the other by loosening of the teeth, but its most prominent feature is tremor.

The former was frequently seen in years gone by, when mercury was excessively used in the treatment of syphilis, and extensively in many diseases, it being considered necessary to give mercury till a "gentle" salivation was produced. The idea of a gentle salivation is given by Grainger in his "Essay on West Indian Diseases." Treating of Yaws he says, "the best plan is to maintain a gentle salivation for six weeks or two months," and immediately adds, "the quantity of spit in twenty-four hours should never exceed a pint."

The latter form, namely, mercurial tremor was also more frequent formerly than at present, the improvement being due in a large measure to mechanical and electrical processes having displaced manual labour, also to more care being taken by those who work with the metal.

*The causes* of mercurial tremor may be divided into: (a) Predisposing. (b) Exciting.

(a) *Predisposing.*

1st. Want of proper ventilation in the workrooms.

2nd. Defective cleanliness on the part of the workers, such as not washing before eating.

3rd. Anything which tends to lower the general health, especially intemperance. In the mercury mines at Almaden, the greatest number and the worst cases occurred in those who drank heavily.

(b) *Exciting.* The chief exciting cause of the disease is inhalation of the vapour of the metal or of its salts. The "trembles" may also be brought on by handling the metal, and less frequently by rubbing in ointments. The actual exciting causes, therefore, exist among:

1st. *Persons liable to inhalation of the vapour*, as in those employed in the recovery of the metal from the ore by heat. The makers of thermometers and barometers, water-gilders, looking-glass silverers, and workers in chemical works. Dr. Legge informs me that there is a danger among workers in the manufacture of electric meters, and of incandescent electric lights where mercury pumps are used to create a vacuum.

During sixteen years at Guy's Hospital, from 1883 to 1899, there were only two definite cases from any of these causes. One in 1886, a water-gilder by trade, the other in 1891, a man who worked in chemical works where mercury was used, and with which he came a good deal in contact.

2nd. *Persons employed in hatters' furriers' work* run the risk of mercurial poisoning both from inhalation of

the fumes of nitrate of mercury, and also from ingestion of particles of fur impregnated with the same salt.

During the latter half of 1900, two patients were admitted into Guy's Hospital suffering from mercurial tremor. Both these patients were engaged in work in the same rabbit skin factory, and stated that a large number of the work people employed, suffered in the same way. Owing to this I was induced to make enquiries.

*Mode in which mercurial poisoning is brought about.*—When mercury is swallowed in the liquid form, it rarely gives rise to poisoning, when absorbed in the form of vapour, it frequently does so. According to Kunkel (12) the inhaled vapour as such, does not pass through the walls of the alveoli of the lungs. It first undergoes condensation and the tiny globules become oxidised and then dissolved. It is difficult to see where this condensation takes place, as the expired air is warmer than the inspired.

The reaction which enables mercury to enter the circulation is not perfectly understood. The perchloride of mercury forms with albumen an albuminate which is insoluble in water, but readily soluble in the presence of sodium chloride. In this reaction probably lies the explanation of mercury poisoning.

*Symptoms.*—The onset of symptoms in industrial mercurial poisoning is slow and insidious in character. Occasionally the symptoms resemble those arising from the long continued administration of mercury medicinally, such as sore gums, excessive salivation, and loosening of the teeth, going on in severe cases to emaciation and tremor. More frequently the onset is quite insidious, nothing sufficient being noticed to trouble the patient until tremor renders him almost helpless.

In hatters' furriers' work the teeth are characteristically affected; they become loose, and certain of them fall out. There appears to be a kind of order in which the teeth are affected. Those which first become loose are the molars in both upper and lower jaws, and these are lost very constantly. Next affected are the canines and incisors, more particularly in the upper jaw, but these

are not so frequently lost as the molars. Such teeth as are left are much blackened and often loose. The blackening is most constant, all the workers in this industry that I have examined present this condition of teeth, and they tell me that practically all the workers suffer in the same manner. Besides the blackening and loosening, the teeth become eroded but not carious, as the enamel suffers more from the acid fumes than the dentine. This was well marked in case 7. Frequently also the gums recede so that the roots are exposed, more particularly of the incisors and canines.

Although the blackening is so constant, salivation does not seem to be very frequent. Cases 2, 3, 4, 5, 6, 7, definitely say they have never suffered from salivation or sore gums, yet they all have the typical teeth, and the only symptom they complained of was tremor. In the gradual poisoning, at any rate in rabbit skin factories, there do not seem to be any signs of gastro-intestinal irritation produced.

The onset of tremors is insidious, and the duration of exposure to mercury fumes necessary to produce them varies much in different people. Some work in a factory all their lives and never suffer, others are affected in a year or two. Case 5 worked in a hatters' furriers' manufactory for twenty-one years as a "carotter," for seventeen years of this time he never suffered any inconvenience, then the tremor commenced. Case 6 worked for only one year at the same work before becoming affected.

The trembling usually begins in the upper limbs. At first the worker notices his hands and arms getting weaker, and less under control, so that he is unable to do any work requiring precision. The unsteadiness is not so great as to prevent him carrying on his work, but a workman assured of his skill becomes shy and nervous, especially when watched. If he removes from his employment now, he soon regains strength and steadiness; if, however, he persists, the tremors get worse, their spasmodic and convulsive character become more and more apparent. It has been proposed to call the tremor an "attention" tremor (13) as it is called forth by the patient's attention being drawn to it.

The tremor affects the voluntary muscles, and so long as the patient is at rest or sleeping does not occur; as soon, however, as

any definite movement is attempted, tremor makes the attempted movement inaccurate. .

The movements are convulsive in character ; if the hand is held out the fingers cannot be kept still, but tremble violently. This movement is not so regular as in paralysis agitans, nor is it like that of chorea, in that the jerking is not so great (unless some definite movement is being attempted), and that between the movements of greater range there is a continuous fine tremor.

In trying to flex the elbow a steady flexion is impossible, in place of this a series of spasmodic jerks occur. This spasmodic movement becomes worse the more the patient's attention is directed to it—for instance, while it is possible to flex the elbow fairly steadily if the hand is empty, yet on trying to carry anything to the mouth, such as a glass of water, the movements become so erratic that the water is spilt or the patient inflicts a blow on his face with the glass. Dr. Pope, in relating the case of a miner at Fruili, says, "he could not with both hands carry a glass half full of wine to his mouth without spilling it, though he loved it too well to throw away" (1).

\* About this time the patient is generally obliged to give up work ; the tremors thereupon improving, only to return again on his resuming the same employment. But if he persists in his work or returns too soon, the tremors become aggravated and gradually extend over the whole body.

The legs are affected next after the arms. During rest the tremor ceases, but returns when any movement is attempted. In Case 2 the patient was unable to move without support ; on standing up the legs began to tremble, and after a minute or so this trembling became so great that she could not even stand without support. The unsteadiness seems to be worse at the knee, the leg jumping about as if hung on wires when it is swung forward to make the next step. In the case mentioned above, after about two steps the legs became so unmanageable that it was impossible for the patient to stand, even if supported.

The jaws are tremulous, making opening and shutting the mouth uncertain. The tongue on protrusion is affected with a fine tremor. The face twitches. The whole head shakes with

a fine tremor somewhat resembling senile tremor. In Case 2 the head was quite still when she was at rest or merely talking, but when she attempted to walk it became very unsteady, probably from the necessity she had of watching her feet. It seemed as if the more attention she paid to her legs the worse her head became.

The tracing of the tremors I have been able to take show nothing characteristic, the rapidity is eight per second, and the amplitude is extremely irregular, becoming worse when any effort is made. Charcot says that a characteristic of mercurial tremor is that it ceases during rest in a remittent manner only, reappearing from time to time without the patient making any movement (11). This is not noticeable in the tracing of the tremor that I have taken.

Involvement of the respiratory muscles is described, accompanied with dyspnœa. Tonic spasms occur frequently attended with pain, called by the Spanish miners at Almaden "*calambres*" or cramps (6).

The stage of tremors alone is not dangerous to life but at a later stage the brain becomes affected, resulting in headache, sleeplessness, loss of memory, and delirium. This condition compels the sufferer to stop work, and even now although recovery may occur, death is the more probable event.

Sensation seems to be entirely unaffected; in the cases I have seen there were no areas of abnormal sensation.

The reflexes are quite normal, as are also the functions of the bladder and rectum. There is no reaction of degeneration. Nystagmus never occurs.

Anæmia is marked in most cases, and is of the ordinary "secondary" type, *i.e.*, both the number of red corpuscles and the hæmoglobin are reduced about equally, so that the colour index remains near 1.

The state of the bowels does not show any tendency to relaxation. All the cases I have seen, say they have never been troubled with diarrhœa. The urine and fæces contain no mercury (Cases 2 and 3).

In women, menstruation becomes scanty or ceases, probably as part of the anæmia. Pregnant women are liable to abort.



In late stages the lungs are affected, cough and signs of phthisis may be present, the morbid changes in the lungs in some cases being the cause of death.

*Morbid anatomy.*—Owing to the rarity of death occurring from chronic mercurial poisoning during recent years, not very much is known about the morbid anatomy. In the few cases which have occurred nothing characteristic has been found. The heart and liver appear to be normal.

Signs of phthisis, such as cavities, are frequently found in the lungs, and in many cases the immediate cause of death is due to these changes.

Kaufmann, quoted by Kunkel (12) says that the changes which occur in the kidneys are the same as those seen in the large white kidney. In the post-mortem report of Case 1, however, there was no mention that the kidneys were either large or white. In this case mercury was extracted from the kidneys, liver and brain. Weight for weight of the organ, more mercury was extracted from the kidney than from the liver or brain. It was therefore shown that mercury was absorbed and deposited in the kidney, liver and brain, and that from them it could be obtained by ordinary chemical means (7).

Prévost has described a case of poisoning by mercuric nitrate, in which the kidneys showed a peculiar form of nephritis. The epithelial cells of the convoluted tubes were granular, and the tubules in places were filled with compact masses of chalk. The same investigator has shown that the ash of calcined bone from a case of mercurial poisoning contains less calcium than normal (8).

Heilborn describes a change in the bones; namely, a lessening of the medulla, and a thinning of the compact tissue associated with increased brittleness (9).

*Pathology.*—The pathology like the morbid anatomy is obscure. The theories are various. Jussieu's ideas was that the tremors are "les tristes effets du séjour du sang dans les vaisseaux du cerveau, devenus variqueux par le poids de quelques particules mercurielles, qui y on sejourne" (2).

Some authors have held the view that the muscles are the seat of the lesion—this, however, is improbable, for there is no

muscular wasting, and the electrical reactions are unchanged. Again, the spasmodic nature of the tremors and later, the development of cerebral symptoms are against this view.

That it is not a neuritis is shown by the absence of "pins and needles" in the limbs, of wrist and ankle drop, or other local paresis, and of pain on pressing the nerves.

That it is not any lesion in the root zones, or in the anterior cornual cells, is shown on the one hand, by the fact that the reflexes are normal, and on the other, that there is no muscular wasting, nor any form of reaction of degeneration.

There is no spasticity, therefore no sclerosis of the lateral tracts. No definite morbid patches have been found as in disseminated sclerosis, in which the tremor is so similar to that of mercurial poisoning.

Senile tremor, and the tremor of paralysis agitans are somewhat like that of mercurial poisoning. In neither of these diseases is the nature of the lesion known, though in the latter it is supposed to be situated in the cerebral cortex.

Charcot (10) does not believe there is a mercurial tremor, and supports his statements by the following facts:—

(a) In acute and sub-acute mercurial intoxication the existence of a tremor has never been proved.

(b) Men may remain for years in a workshop without a case of tremor occurring, but when one does occur a real epidemic sets in.

To Charcot, mercurial tremor is a hysterical tremor, which may exist alone, and without other concomitant symptoms. The patient would then develop a toxic hysteria, like that produced by alcohol or syphilis.

This view is hardly tenable, because in all the cases reported, some long exposure to mercury was necessary before tremor appeared. The action seems to be due to a cumulative effect of the poison. In acute or sub-acute poisoning this effect would not be produced, for as soon as symptoms of mercurialism appeared the exhibition of mercury as a remedy would be stopped.

As to the second fact, brought forward by Charcot, men certainly do work for years without becoming affected—and some workers never are affected—but the testimony of the cases

I have seen is that there are always some workers affected, but not so severely as to need medical help.

Letulle says that in five cases examined by him, three presented hysterical symptoms. In none of the cases I have seen (eight in number) were there any hysterical symptoms.

Again, amongst the workers in the mercury mines at Almaden the most frequently affected were those who did not take care about washing before eating, or who did not change their clothes before leaving work. Those taking precautions scarcely ever suffered.

The mercury molecules appear to enter into some combination with the nervous substance of the brain, thereby interfering with the nervous centres of motion, but leaving those of sensation intact. Dr. Swayne Taylor showed that mercury could be obtained by ordinary chemical processes from the brain of a man who had died from chronic mercurial poisoning. (7).

*Diagnosis.*—The disease of a person who presents himself suffering from tremors will have to be diagnosed from other conditions in which tremors occur; such as Graves' disease, general paralysis of the insane, senile tremor, alcoholism, chorea, disseminated sclerosis, and paralysis agitans.

In exophthalmic goitre the tremor is very fine, and not irregular as in mercury poisoning, also the accompanying symptoms would point to Graves' disease.

Senile tremor will probably not present much difficulty, as the mercury tremor is unlikely to occur in old age. If the patient suffering from mercurial poisoning should happen to be very old, there will most likely have been at least one previous attack. In alcoholism, again, the tremor is very fine and regular, and there will be other symptoms of alcoholism.

Chorea presents more resemblance, the movements are irregular, but they occur while the patient is at rest. In mercury poisoning the spasmodic character only comes out when the patient attempts some definite movement.

Disseminated sclerosis resembles it in that the tremor is an attention tremor, but in this disease nystagmus is an almost constant symptom, but never occurs in mercury poisoning. Charect, quoted by Marie (11), says that the mercury tremor only ceases during rest in a remittent manner reappearing from

time to time without the patient making any movement, whereas in disseminated sclerosis the tremor is completely absent during rest.

In paralysis agitans the tremor is constant, and is associated with a characteristic deformity, the joints being held in a semi-flexed position. The tongue, too, on protrusion is not tremulous.

A tremor brought out by movement, absent during rest, the absence of nystagmus, normal reflexes, and the history of exposure to mercury ought to make the diagnosis clear.

*Prognosis.*—The prognosis is good on the whole. If the patient is removed from work during the early stages, when the teeth are loosening, or soon after the tremor commences, complete recovery will take place in a few weeks. Even if the tremor is so far advanced as to render the patient helpless, complete removal from work and rest in bed restores him to health in the course of two or three months. In the later stage, that of delirium and insanity, the prognosis is bad. I have been able to find only two fatal cases recorded (7 and 4). In the earlier part of the last century a large proportion of the cases died from phthisis. It is recorded that in Paris in 1821 looking-glass workers could not stay at work more than eight or ten years. When obliged to stay too long at work they became pale, intelligence and memory gradually failed; they fell into a kind of idiocy and finally died of consumption. (3).

*Frequency of poisoning in Hatters' Furriers' Manufactories.*—In August, 1900, a case of mercurial tremor was admitted into Guy's Hospital, the patient working in a Hatters' Furriers' manufactory. I went down to the factory and the employers kindly gave permission for me to be shown over the factory and have everything explained to me.

They said they were surprised to hear of a case in the hospital, and they did not know of any other in the factory. Of course I was not allowed to examine the workers. In November another patient was admitted suffering similarly, and also employed in the same factory.

As a result of these notifications, Dr. Legge, of the Home Office, visited this factory and examined the workers, and he tells me that about 60 per cent. are suffering from mercurial poisoning.

At the other factories where I tried to get permission to see

over, it was absolutely refused. At one I was told they had never heard of the "trembles."

The following table which Dr. Legge has drawn up, and kindly given me permission to use, shows the result of his examination, in *eight* different factories, of *one hundred and eleven* people who had worked for one year and upwards.

Process.	Number Examined.			Teeth Bad.			Teeth Fair.			Tremor.		
	M.	F.	Total.	M.	F.	Total.	Per cent.	M.	F.	Total.	Per cent.	Per cent.
Carotting ...	12	18	30	7	13	20	66·6	5	5	10	33·3	—
Other Processes ...	16	65	81	4	23	27	33·3	12	42	54	66·6	21·6

*Description of work in a rabbit skin factory.*—In hatters' furriers' work a dilute solution of nitrate of mercury is used in the preliminary stage of felt hat making to increase the felting properties of the rabbit's fur. Few combinations would be more likely to affect detrimentally the health of the worker, and more particularly the teeth than that of mercury and nitric acid, especially when volatilised by heat, as necessarily takes place in work of this character.

The danger begins with the mixing of the liquid which is used for dressing the skins on the fur side with a view to removing the grease in the fur, so as to increase its felting properties. The liquid used is a solution of nitrate of mercury; the proportion of mercury to nitric acid varies in each factory and is regarded as a trade secret.

"Mixing." The mixing of the mercury and nitric acid is generally done by a man, either out of doors or in a room set aside especially for the purpose. The liquid is served to the workers mixed with water ready for use. The liquid is prepared in some factories once a week, in others every day.

"Carotting." This is the technical name for the process by which the liquor is applied to the skin. There are two forms, "hand carotting" and "machine carotting."

"Hand carotting." In this process the worker scrubs the skins with a hard brush dipped in the solution contained in bowls at his side. The skins are placed on an earthenware slab, which, in the factory I saw, slanted away from the worker and was connected with a waste-pipe. In some factories I believe a plain wooden slab is used with no special means for the removal of the fluid. In the factory I saw, the workers wore indiarubber gloves, extending as far only as the wrists. In some places indiarubber finger-stalls only are used. In any case the arms and clothes are splashed with the solution. In some factories men only are employed on this work, as it is considered unfit for women, being both hard and dirty. Both men and women are employed in this work at the factory where cases 2, 3, 4, and 5 occurred.

"Machine carotting." In this process also there is direct contact between the worker and the poisonous liquid. The undressed

skins are placed between rollers, and thrown out dressed by mercury nitrate. Each skin having passed through the machine is removed by hand in a wet state.

"Drying." The skins are next removed to stoves for drying. They are placed on racks in a steam-heated chamber, which is entered by the people who take the skins in and remove them. In the factory I inspected the chamber was in two stories. The upper was reached by a flight of stairs on the outside. I was told there was very little danger to the worker if the chamber was properly ventilated. In chambers of this type the workpeople are exposed to the double danger of poisoning from handling the dressed skins, and also from inhalation of the vapour of mercury nitrate.-

In some factories the skins are placed on "horses" which slide in and out of the chamber, so obviating the necessity of the worker entering the dangerous atmosphere. Special ventilation is also sometimes used for removing the dangerous fumes as they arise.

"Tying-up and damping." If the skins are not at once required for further processes they are tied up and stored. After being stored for a short time, it becomes necessary to damp them in order to soften them for the cutting machine. This is generally done by girls, who sprinkle the skins with water. The skins so handled are of course impregnated with nitrate of mercury. The people who are employed on this work frequently show signs of poisoning, as loose and blackened teeth, for the process is carried on in the ill-ventilated place used for storing the skins.

"Cutting." The next process is removing the fur from the skin. This is done by a very ingenious machine with rotating knives. The skin with the fur on it is "fed" into the machine, in which the fur is shaved from the skin, this (the skin) falls in narrow shavings into a box below, and is afterwards used in making glue. The fur is delivered into a tray, in the form it went into the machine, *i.e.*, it looks as if it were still adhering to the skin. The worker, known as the "locker," who stands at one side of the machine to receive the fur, removes the outer edges, leaving only the fur of the back, which he compresses

with his hands and places in bags ready for transmission to the hat manufacturer. This fur is impregnated with mercury nitrate. A specimen gave on analysis 1.34 per cent. of mercury nitrate. The large majority of workers at the machines are women. Considering the nature of the work in the machine room it is wonderful that more fluff and dust are not apparent in the atmosphere.

The degree of danger in this industry is dependent on:—

(1.) The conditions under which the "mixing," the "carotting," and the "drying," are carried on, and the state of the machine room.

(2.) The susceptibility of the worker.

*Treatment.*—The treatment is both preventive and curative. In the hatters' furriers' processes the preventive treatment should be of three kinds.

(1.) Means ought to be taken to limit as much as possible contact of exposed skin to the liquid, the worker to nitrate of mercury fumes, and to dust and fluff impregnated with the same compound.

The chief danger is in mixing the liquor, in carotting, in the drying room, and at the machine. The mixing should be done when the other work is not in progress, and in a place well ventilated and especially constructed to carry away the fumes.

In carotting, precautions should be taken to prevent the worker's hands from coming in contact with the solution. This may be done by using forceps for holding the skin while it is brushed, or better, by wearing indiarubber gloves coming well up the arms, care being taken that the gloves are always in good repair. The carotting should be done on a sloping bench, so that the surplus liquid can run into a channel ending under a flue, up which is a forced draught, all fumes being thus removed from the carotter.

The drying chamber should be provided with racks which are made to slide in and out, in place of the old fixed racks in the chamber. By this means the necessity for entering this most dangerous room would be removed.



In the machine room, the upper part of the machine ought to be enclosed. The lower part, into which the shaved skin and most of the dust falls, might be made to fit tightly to the machine, and connected with a shaft in which is a fan to remove the dust. The workers at the machine ought to wear overalls and a covering for the hair, so that on leaving work they carry no nitrate of mercury with them.

(2.) Means ought to be taken to ensure personal cleanliness on the part of the worker. This involves co-operation between employer and employée.

In other dangerous processess it is compulsory for one hand basin to be provided for every five persons. This should be enforced in this industry. The basins to be provided with a waste pipe, hot and cold water. Soap, towels, and nail-brushes should also be supplied. Every worker ought to wash, remove overalls and head gear before eating, or leaving the factory. No food should be taken in places where any process of treatment of dressed skins is being carried on.

(3.) Age limit. In Belgium, by the law of April 15th, 1898, no person under sixteen years of age may be employed in a factory where skins or fur are treated with nitrate of mercury. In France, by the law dated May 13th, 1893, no woman or child may be employed in these factories. I am not aware that there is any corresponding protection in this country.

*Curative.*—The most important point is removal of the sufferer from his work at once ; unless this is done it is useless to attempt a cure. The patient should have complete rest in bed and be fed on nutritious food.

The drug most relied on is potassium iodide. It is said that it must be given in small doses, as large doses have the power of dissolving the mercury in the tissues (5), the result of this being that symptoms of acute poisoning may arise (14).

Oliver recommends phosphide of zinc in doses of one-tenth to one quarter of a grain three times a day (15).

Sir William Gull had some satisfactory results from the employment of galvanism (16).

The only absolute cure is complete and permanent removal from the work.

## LIST OF CASES.

Owing to the difficulty thrown in my way by some of the work people themselves, I have not been able to get even slight reports of some of the cases of which I know. Those people I have seen who are still employed at the factory, have begged me not to mention to their employer that I have seen them, as they would be immediately dismissed. Two cases not mentioned here, as soon as they found out what I had called for absolutely refused to tell me anything.

CASE 1.—R. B., æt. 32, Furrier (Guy's Hospital Reports, Vol. x., 1864, p. 173).

*Admitted* into Guy's Hospital, December 10th, 1863.

*Died*, December 25th, 1863.

*History*.—Patient is married and has four children. Four years ago he began his present work; previously he was a butler. His present work is packing skins which have been treated with mercury nitrate and afterwards dried. For three years patient did not suffer much inconvenience beyond some weakness, and salivation during the first three months after starting work, which soon passed off. Twelve months ago he could not hold his hand steady enough for shaving, soon after he lost complete control of his limbs. He gradually lost voluntary control of his limbs when moving or standing, and three or four months ago began to twitch in bed, one month ago he gave up his work.

*On admission*.—He could walk fairly well with a little help, but when standing could not control his limbs, which trembled considerable and even slightly when lying in bed. He feels very exhausted as he has had no sleep for several nights. His head aches. Tongue furred, bowels costive, sweats profusely. Heart-sounds cannot be heard because of the spasmodic movements of the muscles of the chest. December 12th: Patient became delirious during the night, getting out of bed several times. The delirium continues to-day. December 14th: Continues delirious. December 25th: Patient has been gradually getting weaker and taking no food. He died to-day.

*Post-mortem*.—The body appeared quite healthy, it was well nourished, and there was no wasting. The muscles were red and healthy looking. No lesion to be found anywhere by naked eye.

*Chemical analysis*.—Urine soon after admission was shown to contain no trace of mercury. Six ounces each of brain, liver, and kidney, were taken and dried, half the residue was digested with the following solution:—Pure hydrochloric acid, one part, and distilled water, four parts, concentrated by evaporation. A spiral of thin gold foil wrapped round zinc was put into the solution while warm and left for twenty-four hours. Kidney: The gold placed in the fluid after digesting with kidney residue, had a whitish tint, the zinc was dissolved. The gold foil was washed, dried, and heated, in a reduction tube, when globules of mercury were obtained. Brain and Liver: The gold

which had been used in the fluid from these two organs gave no deposit of mercury. The other halves of the residues were tested with copper instead of gold, when the kidney gave globules of mercury  $1/1750$  inch in diameter. The brain and liver each gave globules  $1/2600$  inch in diameter. It was therefore proved that mercury was absorbed and deposited in the organs of the man, and could be obtained by ordinary chemical processes from three ounces of each organ used, but that weight for weight kidney yielded most. Also in reference to tissues, copper separated mercury more perfectly than gold and zinc did galvanically.

CASE 2.—J. P., æt. 50. Machinist in rabbit-skin works.

Admitted, August 8th, 1900, into Guy's Hospital.

Discharged, October 30th, 1900, much relieved.

*History*.—Patient has been doing her present work for over twenty years, she has been perfectly healthy until two years ago, when she began to get tremulous. She has never had any illness. She says her work involves living a great part of her time in an atmosphere filled with "vapours of mercury and various acids." Several of her companions are involved in the same way.

*On admission*.—When brought to the hospital she was unable to walk by herself, she takes a few steps and then falls down, her legs giving way under her. If trying to walk her whole body trembles, and also her head nods with a very fine tremor. When lying down or sitting up in bed there is nothing at first sight to indicate what her complaint is. Her hands are quite still and the expression of her face is one of comfort. She is a fairly well nourished woman with a healthy colour. *Nervous system*.—Head kept quite steady while at rest in bed, or sitting quiet. It commences to tremble with a very fine tremor on the slightest exertion. Lips tremble when she is asked to speak. Tongue tremulous when protruded. Pupils react to light and accommodation. Optic discs quite normal. No nystagmus. Her voice has been affected lately, getting husky, but her larynx is quite normal. *Arms*.—There is a continuous tremor on attempting any voluntary movement, making feeding and writing quite impossible. The tremor disappears entirely when she is at rest and during sleep. The tremor is not of the arm only, but also all the fingers tremble. *Elbow-jerks* normal. There is no tenderness of the muscles or of the interdigital nerves. *Legs*.—The legs like the arms become very tremulous on attempting any movement, such as walking, making it quite impossible without support. *Knee-jerks* normal. No ankle clonus. There is no tenderness in the calves, though the interdigital nerves in the first space of both feet are tender on pressure. *Sensory*.—The sensation is perfectly normal all over the body, legs and arms. No reaction of degeneration. Bladder and rectum both normal. *Alimentary system*.—Tongue tremulous but clean. Teeth very black, and the molars in both jaws are lost. No salivation in excess and there never has been. Gums not tender and have never been sore, there is no blue line. Abdominal organs all healthy. *Circulatory and respiratory* systems normal. *Urine*.—Light yellow. Specific gravity 1018. Acid in reaction. Blood, albumen, sugar, pus absent. Very carefully tested for mercury by evaporating and placing strips of copper in the fluid, none found. The fæces were also tested for mercury with a negative result. *Treatment*.—Patient was kept in bed and fed on full diet. No drugs were administered at first. She gradually improved, and in a fortnight could feed herself. During the last four weeks she was in hospital she took five grains of potassium

iodide three times daily; the improvement went on, but did not seem to be any more rapid. The urine and feces were tested periodically while she was in the hospital, no trace of mercury was at any time detected. Discharged on October 30th, to a convalescent home and told not to return to her former work. On discharge there were no signs of tremor unless the patient became excited, when slight ones were noticed.

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CASE 3.—M. M., æt. 52, Locker in rabbit-skin works.

*Admitted*, November 16th, 1900, into Guy's Hospital for mercurial tremor.

*Discharged*, December 6th, 1900.

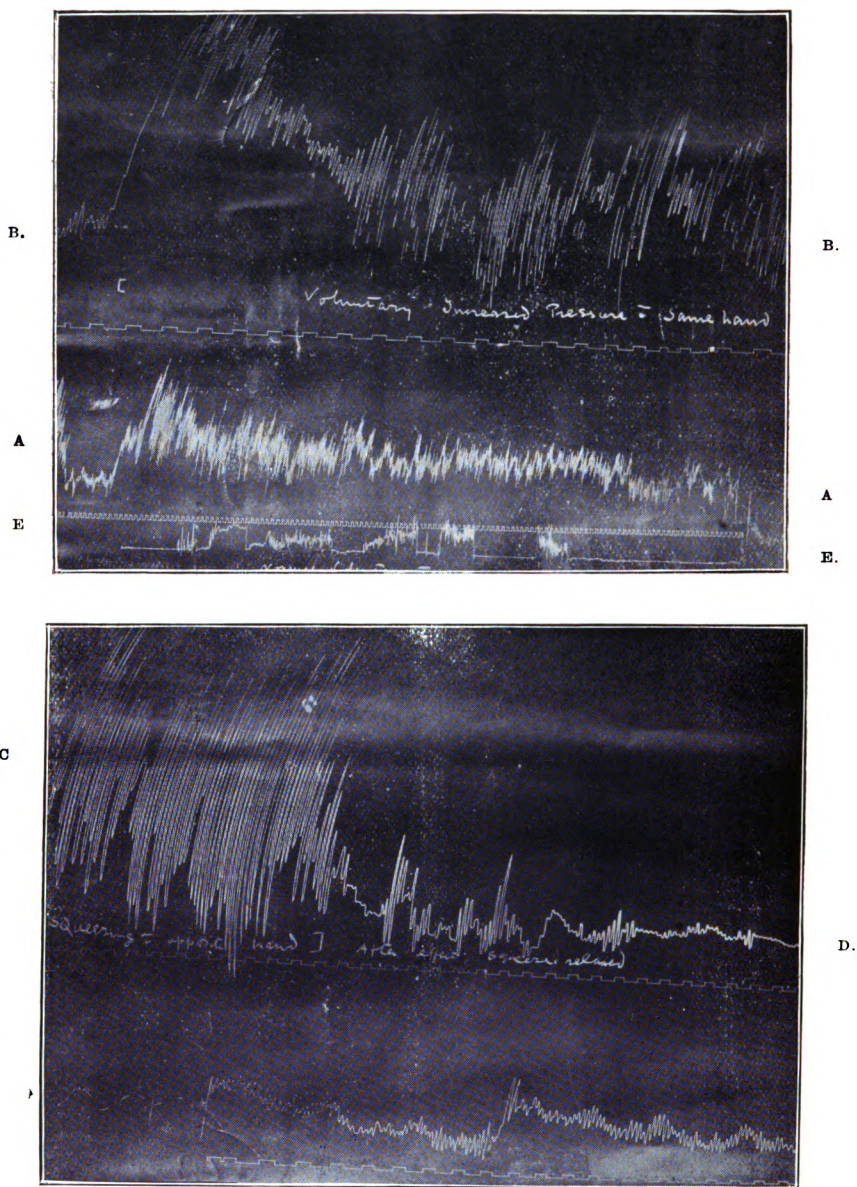
*Family history*, unimportant.

*History*.—Patient has always lived in London, and has worked at her present occupation since she was ten years of age, that is, for forty-two years. This work consists in collecting the fur which has been removed by machinery from rabbit-skins, which have been previously treated with preparations of mercury. She works in a large warehouse, which is well ventilated by skylights which are open night and day. Patient says that all the work-people are obliged to change their clothes and wash before leaving. She always washes her hands before eating. The dust from the work is very thick at times. She is a married woman, and had one child, who died at the age of thirteen of "cold and dropsy." She has never had any miscarriages. Patient has never been anything but a moderate drinker. *Previous diseases*, nil. *History of present illness*.—For some years patient has been subject to attacks of "trembling" at times; but they have always gone off soon. The last attack came on suddenly about four weeks ago, with pain in the back, shivering and headache. Patient could not stand, and was sent to the hospital after a few days, in a cab. Since then till admission she has been getting gradually better, and the trembling has decreased a good deal.

*Condition on admission*.—Patient is rather an anæmic woman. Her teeth are black, and her breath is foul and metallic smelling. She has a slight cough. She is deaf, and her voice is rather tremulous. Her hands show marked tremors, especially so when she tries to pick anything up. There is no ulceration of the mouth. The tongue is tremulous, but of good colour. Her legs and feet also tremble. *Nervous system*.—*Head*, no tremor; *lips*, no tremor; *tongue*, tremulous; *pupils*, normal; *optic discs*, normal. *Arms*.—There is a continuous fine tremor on attempting to use the arms, but it is not so great as to prevent the patient feeding herself. No tremor while asleep or at rest. There is no tenderness of the muscles. *Elbow-jerks*, normal. *Legs*.—There is slight tremor of the legs, but it is not sufficient to prevent walking without support. *Knee-jerks* present, but not very brisk. There is no tenderness of the muscles. *Sensory*.—Sensation is perfectly normal all over the body, arms and legs. No reaction of degeneration. Bladder and rectum both normal. *Circulatory and respiratory systems* normal. *Alimentary system*.—Tongue tremulous, but clean; teeth much blackened. No ulceration of gums, and there never has been. Lips, anæmic. Abdominal organs all healthy. There is no history of colic at any time. *Urine*.—Albumen, sugar, and blood absent; acid in reaction; specific gravity, 1020; there was no trace of mercury. The urine was tested regularly while in the hospital, and no trace of mercury was at any time found. The feces were tested regularly, and no trace of mercury was ever found. *Blood-count*.—Red corpuscles.



*Chronic Mercurial Poisoning, with special reference to the Danger  
in Hatters' Furriers' Manufactories.*



Tracing of Tremor in Case 3.

3,800,000; white corpuscles, 10,000. 16th November, 1930: Patient ordered five grammes of potassium iodide three times daily.

The patient gradually improved under this treatment, and was discharged cured on December 6th, 1900.

*Explanation of tracing on opposite page.*

- (A.) Right arm resting quietly. Six tremors per second, of much greater amplitude and regularity than a normal voluntary tremor, which was only four per second.
  - (B.) Voluntary pressure of finger on tambour. Seven tremors per second. Amplitude about seven times as great as A.
  - (C.) Strong voluntary movement (pressure) of hand not on tambour. Six tremors per second. Amplitude about twelve times as great as A. The excursions very much more regular than any of the others.
  - (D.) Immediate after-effect of C. Five tremors per second. Very regular in amplitude, becoming very small indeed after a short time.
  - (E.) Normal voluntary tremor.
- (Published with Dr. Fawcett's kind permission).

CASE 4.—S. T., æt. 45, Married. This woman is a "locker" in the same factory as cases 2 and 3. She has worked there for about fifteen years. She is now very tremulous. She is quite unable to guide her hand with accuracy, and in trying to carry the candlestick to light me downstairs, it was held so very unsteadily that I had to take it from her. Her legs, too, are very unruly, and in walking she has to take hold of objects to prevent herself falling. Her head is unsteady and the tongue tremulous. When I went into the room, she at first showed no signs of tremors, but after talking to her for a minute or two they came on, and never left her while I was there. The reflexes are normal, and as far as I could ascertain sensation is perfectly normal also. There is no nystagmus. She is anæmic. She has never had any salivation or sore gums. Her teeth are very black and she has lost all her molars, and the roots of the incisors are exposed. I was unable to obtain a specimen of her urine. She absolutely refused to come up to the hospital to be more carefully examined, for fear it should get to her employers knowledge, and she would be dismissed from her work.

CASE 5.—T. C., æt. 57, "Carotter" in rabbit-skin works. This patient at the present time has no tremor, and has had none for three years. For twenty-one years he worked at carotting; during the first seventeen years he never had any trouble, he then began to become tremulous. His tremor was so bad he was unable to feed himself, and he was on three occasions admitted into a hospital. He says most emphatically that he never has had sore gums or salivation. His teeth are very black, he has lost the two molars in upper jaw. Since the last time he was in hospital he has been employed on different work, so as not to be exposed to any danger of mercury poisoning.

CASE 6.—M. M., æt. 22. Married. For two years this patient worked in a rabbit-skin factory. During the first years she was doing fur pulling, i.e., she was working at the skins before they were treated with mercury. During this year she was quite well. She was next moved to work at the brush, she only worked at this for one year. At the end of that time she had sore gums, and her teeth were getting loose, and she says she was næmic. She saw a

doctor, who advised her to leave her work, which she did, and has had no more trouble. At the present time she has no salivation, her gums are quite right, and her teeth are not loose. Her teeth are slightly blackened and she has not lost any, otherwise they appear normal. She has had two children, no miscarriages. She has always menstruated regularly.

CASE 7.—G. S. æt. 25. Married. This patient worked for one year only at the machine as a feeder. She was never employed at any other process in the factory. At the end of the year she suffered from tremor very severely, and could not hold anything. A doctor advised complete removal from the work. She followed this advice three years ago, and is now apparently quite well, though she says she still trembles when excited. There was no tremor visible while I was talking to her. She has never had salivation or sore gums. The teeth are blackened, and the upper and lower molars are lost. The gums recede on the incisors and canines. The upper left canine shows the effect on the enamel, which has disappeared on part of the anterior surface, and between it and the premolar next to it. She used to suffer much from anæmia, with shortness of breath, palpitation, &c. Menstruation was irregular. She had one child, born dead, after she left the work.

CASE 8.—E. E., æt. 23. This patient worked for one year at a machine in a rabbit-skin factory. She then became slightly tremulous, and her gums became sore. She has not lost any of her teeth, though they are slightly blackened. She was anæmic. Twelve months ago she gave up work, and has now lost the tremor, and is not anæmic.

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# THE SYMPTOMS IMMEDIATELY PRECEDING DEATH FROM EXOPHTHALMIC GOÎTRE.

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By A. J. CLEVELAND, M.D.

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AN examination of the fatal cases of Exophthalmic Goitre admitted into the Medical wards since 1887 shows that although a great similarity exists in the group of symptoms shortly preceding death in each case, yet there is no single one which makes an unfavourable ending certain, and there is considerable difference in the extent to which the various symptoms may be marked. In all, 84 cases of the disease have been admitted, 9 being males and 75 females. Of these, fourteen have died, all women, but of these fourteen one was clinically a case of mitral regurgitation with all the evidences of failure of compensation; in another the patient had severe diabetes and the Graves' disease was slight, and a third was complicated by acute rheumatism, pericarditis and mitral stenosis. Omitting these three cases, eleven uncomplicated ones are left, giving a mortality among the female cases of 15 per cent. Naturally only the worst examples of the disease are admitted into the hospital, and the further history of those discharged is not recorded, so this figure is of no value as an indication of the death rate.

To the eleven cases in our Medical Reports I have added two others which were admitted into the Surgical wards under Mr. Jacobson. Two of the thirteen cases were operated on, but they each died so soon after the operation, with symptoms so like the uncomplicated ones, and without there being any evidence of other cause of death that I have included them.

The train of symptoms which is common to all the fatal cases consists of a general aggravation of all those usually found in this disease, though the mental change is perhaps the one most strikingly altered. Thus, although normally these patients are neurotic and excitable, in all the fatal cases except the first the mental condition was one of active delirium, often requiring restraint. With this, sometimes preceding it, and sometimes following, the temperature was raised often to 103° and 104°, the pulse became very rapid, and the breathing rate increased. Vomiting and diarrhoea were generally present, often occurring before the other symptoms. Sweating, dermatitis, jaundice, and in one case a convulsion, were also noted.

The patient is apparently the victim of a general nerve storm, which, although affecting some centres more than others in different cases, yet affects all to a great extent. It is not easy to make out what it is that starts this nervous storm, or to see by what signs its advent can be foretold. The early appearance of a profound mental change suggests that some nervous or physical shock is the exciting cause.

In Case 13 the patient's husband ascribed her delirium to the sight of a patient being carried away for an operation, while Cases 3 and 4 shewed great mental excitement almost immediately on admission after a long journey. Possibly in some stages of the disease the sudden change of surroundings occasioned by admission into a general ward may be sufficient to upset a mind already badly balanced.

This alteration in the mental faculties was usually described in the reports as "Delirium," the patients being restless, getting out of bed, or "wriggling about the bed," and generally refusing nourishment, the condition being one of severe hysteria. Delusions and hallucinations are not mentioned; one patient

was melancholic, gradually lapsing into a state of complete lethargy, though at intervals she became restless. Thus, in these cases the higher intellectual centres seem to be the starting-point of a nerve storm, which soon involves the lower ones. An analogous instance is found in the onset of coma in diabetes. Here mental or physical excitement may undoubtedly bring on a fatal coma.

The result in the two cases operated on can be explained in two ways. One is that the nervous system was upset by the direct interference with the nerve-supply of the thyroid gland, just as it may have been by some mental shock in cases 3, 4, and 13; the other that the incision and removal of part of the gland allowed the rapid pouring out of the secretion to an absorbing surface, and thus caused a sudden access of the fatal symptoms. This latter was suggested by Putnam (Brain, Part ii., 1894), who also quoted a case of Rehn, where death from cardiac failure followed shortly after an operation consisting only of ligaturing the thyroid arteries, and he says "that there was therefore in this case no reason for accusing thyroid poisoning of being the cause of death." Still, this explanation supports the view, which is, I believe, the one most generally favoured, that the disease is due to a hyperthyroidism.

Osler, in his "Principles and Practice of Medicine" strongly supports this theory by a comparison of this disease with myxœdema, and also draws attention to the aggravation of exophthalmic goitre by the administration of thyroid extract. Whatever be the true pathology of Graves' disease, whether the fault be primarily in the thyroid gland or in the big nerve centres of the brain, it is evident from clinical observation that it is through the nervous system that the patient suffers, and there appears to be a stage in the disease when the nervous system becomes very susceptible to any disturbing factor, though from the cases I have examined there seems to be nothing to indicate when this stage was reached. Some cases have benefited from an operation, so the toxicity of the secretion must vary independently of the symptoms of the disease.

Case 5 would certainly seem to have been a favourable one for operation as, though the disease was well marked, the patient was in a good, general condition; yet within a few hours of coming round from the anæsthetic she was the victim of the fatal nervous storm.

Alteration in the respiration, both as regards rate and quality, was a marked feature of some cases; in four attended by a good deal of cyanosis and distress, for some of which oxygen inhalations were administered. The most simple explanation of this symptom would be that it was caused by the pressure of the enlarged thyroid on the trachea, and I believe that this is how the severe choking sensations these patients so often complain of are generally accounted for. If this were the case, one would expect to find some post-mortem evidence of the pressure by an alteration in the shape or direction of the trachea itself. In only one of the fatal cases is such a change noted, and in some of them the report expressly states that the trachea showed no signs of compression, notably in case 8, where the patient had been blue and livid, and breathing rapidly. It appears therefore that the respiratory disturbance must be brought about by an excitation of the respiratory centres, and is not due to any local mechanical interference, and this view receives support from the evidences found microscopically in the brain in the only two cases which were examined to that end in our series. Mannheim found in the medulla oblongata in several cases organic changes, which he considers to be the cause of the disease. (*Der Morbus Gravesii*, Berlin, 1894).

The character of the breathing was often peculiar, being rapid and shallow, and was generally, where special note was made of it, described as "catchy;" in Case 8, Dr. Perry gave a bad prognosis on this account.

In five cases in which the respiratory changes were marked the lungs were examined after death and found healthy, so that this symptom cannot be accounted for by pathological changes in these organs themselves, although in one other case there was some broncho-pneumonia, and in another clinical evidence of

lobar-pneumonia; yet in this last case the changes in breathing preceded by several days the apparent onset of the pneumonia.

A very interesting complication or symptom of these fatal cases, and one not easy to explain, is pyrexia. Unfortunately, in some cases, no proper chart of the temperature was kept, while in others the patients had sore throats or pneumonia, which might have explained it.

Leaving out those cases in which there can be any possible doubt, six had considerable elevation of temperature, although nothing was found which could account for it. In one case there was hyperpyrexia ( $105^{\circ}$ ); but I have not included it in the six, as at the autopsy the lung was said to be "congested," but there seems little doubt from the report that the congestion was not pneumonic.

In the two cases operated on the rise of temperature occurred within a few hours of the operation, and can have been due to no septic process.

In most cases of exophthalmic goitre the temperature is a little above normal,  $99^{\circ}$ , and even  $100^{\circ}$  being by no means unusual, and so in the fatal cases there seems to be the same exaggeration of this symptom as of the mental, cardiac and respiratory.

In this connection it is interesting to note that Lorrain Smith has found that in animals from which the thyroid body has been removed, exposure to variations of external temperature shows that the mechanism presiding over heat loss is deranged (*Journal of Physiology*, Vol. xvi., 1894).

Possibly in Graves' disease there is a similar derangement of the heat centres tending to produce an abnormally high temperature.

The tachycardia, so constantly met with in almost all cases, has not been explained satisfactorily, and although it was marked in all the fatal cases, yet it seems to be of little importance as an aid to prognosis.

The symptoms, therefore, which, when occurring simultaneously, indicate that the disease has taken a very unfavourable course, are briefly:—Delirium, generally characterised by hysterical outbursts and great restlessness; rise of temperature, quickened

respirations and pulse, and sickness and diarrhœa. Any of these may occur alone, or sometimes two or more together, and the patients get well. Yet when all are noted the termination is almost invariably fatal.

Naturally it is impossible to verify this conclusion without finding out if the above group of symptoms has occurred and the patient has recovered, so I examined the reports of all the cases of Graves' disease admitted since 1887. In only two does the patient appear to have become "delirious;" in one of these the temperature rose to 101° and the general condition became similar to that seen in the fatal cases, but recovery followed, while in the other although there was delirium, sickness, diarrhœa and a rapid pulse, yet the temperature did not rise and the patient soon got better. Another patient had rapid pulse and respirations with a temperature of 101° to 102°, yet her mind remained unaffected, and she went out a few days later with the pyrexia, etc., persisting, so she cannot be said to have recovered from the acute symptoms, and there is no further record of her.

Many reports note that the patient was "neurotic," "excitable," "easily startled," and in some cases the temperature occasionally rose above 100°, but in only the one case mentioned did recovery follow the onset of serious mental and physical disorder combined; this patient recovered of her acute symptoms and left the hospital in the same condition as on admission, and there is no note of her subsequent history.

Of the other symptoms and complications of Graves' disease there is no evidence in our reports that the prognosis is altered by their presence or absence. Taking them separately, one of the commonest is *vomiting*. This is of frequent occurrence in all cases. Many of the slight cases give a history of vomiting sometimes over long periods and often in the early morning. When this becomes as severe as in Case 10, where uncontrollable vomiting lasted for five days, it is undoubtedly serious, but even in this case there was an interval of several days between its cessation and the onset of the fatal symptoms, so that it does not appear to be of any grave significance.

The same remark applies to *diarrhœa*, to attacks of which these patients seem liable. It is interesting to note that in five of the post-mortem reports evidence was found of congestion of the whole or parts of the gastro-intestinal track. *Severe pain* in the chest or abdomen is a common complaint, being situated frequently in the region of the liver, but it has no relation to either sickness or intestinal disturbance.

*Wasting* is generally a feature of any moderately severe case. In some, weight was lost very rapidly, this being so in most of the fatal cases. That a rapid loss of weight can occur without the course of the disease becoming very unfavourable, was shown by a girl under the care of Dr. Taylor at the end of last year, who lost two stone at the rate of four pounds a week, and a couple of months later was picking up in every way. Some other cases also went out better or no worse, in spite of considerable loss of weight.

Tremors, exophthalmos, the movements of the eyelids and the size of the thyroid, seem to bear no relation to the course of the disease.

The *rate of the pulse* varies very much; in some cases which did well it was extremely rapid on admission, and it was 100 or less in slight cases, while in all the severe ones it was high; but taken alone it is of little value.

*Palpitation* and uneasiness or pain in the region of heart and neck are what seem to affect the patient most, and in those cases which improved it is on the alleviation of these symptoms that most stress is laid, and galvanism and the application of cold are the most beneficial agents in this respect. Otherwise the treatment, whether by *digitalis*, *belladonna* and bromides, or by thymus, or thyroid or suprarenal extracts, has not proved satisfactory.

Two of the fatal cases were *jaundiced*. In no other case did this complication appear, so judging from our own records it is a sign of very ill omen. At neither autopsy was anything found to account for it.

*Pigmentation of the skin* is an interesting complication, but it has no apparent relation to the severity of the disease. As to its

cause it would be useless to conjecture, but the figures show that in the fatal cases it occurred twice, and in the non-fatal it is mentioned eight times. In some patients it was very noticeable, being of a fairly general distribution, in others it was limited to the eyelids, nipples, axilla, etc. In Case 2 patches of leucoderma were found at parts subjected to pressure by garters and corsets.

*Other skin affections* are not uncommon, sweating, flushing, and urticaria may be often seen, and I have known the latter to be brought out by merely drawing the fingers across the patient's chest.

In Case 10 there was a severe dermatitis attended by a good deal of itching and desquamation.

Case 9 had an erythematous rash over the legs which lasted for some days.

*Albuminuria* often occurs ; it is generally slight, as a rule not persistent, and seems of little significance. In none of the fatal cases were any structural changes found in the kidneys.

*Menstruation* is irregular in nearly all the cases, periods of amenorrhœa lasting sometimes six months being the usual abnormality.

*Of associated diseases* diabetes is of considerable interest, but is not common in those cases in which the Graves' disease is active, only two of the cases I have examined were thus complicated ; but a fatal case of diabetes recently in the wards had suffered from Graves' disease some years previously, so probably it would be necessary to analyse cases of diabetes to show the association of the two, as those in which the Graves' disease was latent, or had disappeared, would not find a place in our index under exophthalmic goitre.

*Rheumatic affections* are the most frequent of associated diseases. Mitral regurgitation was diagnosed five times, though in none of the uncomplicated fatal cases was a lesion of the valves found, so perhaps it is not right to conclude that the incompetence was due to an old endocarditis ; probably enlargement of the left ventricle from over action causes a leakage of the valves.



*Acute rheumatism* occurred three times; once alone, once with lobar pneumonia, and once with pericarditis and mitral disease. One case had pericarditis alone. Raynaud's disease was seen in one patient who had also mitral regurgitation.

*Chorea* was diagnosed in one case, but the movements had been present continuously for ten years, so the chorea was not of the ordinary type of that disease.

*Phthisis* and *diabetes* each occurred twice. Case 13 had a healed gastric ulcer.

Counting the two fatal cases from the surgical side, of the 86 cases I have referred to, only 14 shewed evidence of any other disorder, so that it is the rule for Graves' disease to be uncomplicated.

An examination of the post-mortem reports does not reveal any changes common to all or even a majority of the cases.

Autopsies were performed in eleven of the thirteen fatal cases, in four of which the brain was not examined. Changes were found in the brain three times; in one a small recent hæmorrhage between the optic thalamus and the caudate nucleus, which the demonstrator thought was of no pathological significance. In Case 1 changes were found in the floor of the fourth ventricle, and in Case 9 there was thrombosis of some of the branches of the middle cerebral artery, with acute softening of the parts supplied, and also similar changes in the pons. As far as I could ascertain, these were the only two cases which were examined microscopically, and the changes found agree with those described by Mannheim, and also explain well the central origin of the disturbances suggested by the clinical symptoms.

*The thymus* was enlarged and persistent in seven cases, the *gastro-intestinal tract* was congested in three, in one containing blood, and the solitary follicles and Peyer's patches were enlarged and prominent in two others.

Broncho-pneumonia occurred once, and in one case subpleural hæmorrhages and oedematous lungs were described.

*The kidneys* were found enlarged twice, but no structural alteration was observed.

*The spleen* was enlarged in one case.

What strikes one as curious with regard to this disease is that patients suffering from it, even with the signs and symptoms well marked, seem able to recover well from other severe illnesses. Thus, acute rheumatism, pericarditis, and lobar-pneumonia occurred in patients who got well, while Case 7 gave a history of having had pneumonia and nephritis, with delirium. In this respect Graves' disease is in striking contrast to diabetes.

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## ABSTRACTS OF FATAL CASES.

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**CASE 1.**—Rebecca M., æt. 31, single, admitted into Mary ward under Dr. Pavy on September 18th, 1888, for Graves' disease (Pavy, 1888, Report 54). The illness started in February, 1886, with diarrhœa, for which she was treated. In October, 1887, she consulted a doctor for the swelling in neck. She has wasted considerably and for ten days before admission has had great sickness.

*Condition on admission.*—Thin, intelligent woman, with signs of disease well marked. Pulse, temperature and respiration not noted till September 21st, when temperature 102° and pulse 140. Sickness had not continued and diarrhœa was better. September 29th, temperature 103°, pulse 140, respiration 48. Great pain in lumbar and iliac regions. October 1st, jaundice of conjunctiva. October 2nd, died. No mention made of mental condition, and the report is very sparing of details. The temperature remained raised from September 21st, and the pulse increased in frequency and at times became very feeble.

*At the autopsy.*—Body extremely emaciated (fifty-three pounds), universally jaundiced. *Brain.*—Capillaries of anterior part full and especially congested at the floor of the fourth ventricle. No hæmorrhages. Microscopically there were inflammatory changes with hæmorrhages beneath the floor of the fourth ventricle. Thymus easily recognised. Thyroid enlarged. *Lungs* showed broncho-pneumonia. *Other organs* normal.

**CASE 2.**—Maud P., æt. 24, single. Admitted into Clinical, under Dr. Taylor, on November 23rd, 1887. (Clinical, 1888, Report 45). *Family history.*—Mother a confirmed invalid, and subject to "fits," with a history of consumption in her family; rest of family healthy. *Present illness* began fifteen months ago with weakness and breathlessness. Eight months ago thyroid noticed to be enlarged.

*Condition on admission.*—Thin, nervous and excitable. Thyroid enlarged, exophthalmos marked. Skin universally pigmented, with patches of leucoderma at points of pressure. Pulse 132, temperature and respiration not noted. Treated with digitalis and iron. She progressed well, and on

December 27th galvanism was tried to the neck. January 4th, 1889, complains of pain in hepatic region; is hysterical, temperature 99°. January 6th, pains all over, temperature 102°. January 10th, has been vomiting, urine normal. January 15th, sickness continues; wasting rapidly; is jaundiced. January 17th, diarrhoea noted. January 21st, pulse 130; is very hysterical. January 22nd, has wasted greatly; mental condition very unstable. She died quietly on January 23rd, at 2 a.m. Jaundice seems to have persisted. No record of pulse, temperature, or respiration beyond that given.

*At the autopsy.*—Brain healthy. Thymus enlarged. Lungs, heart, kidney and liver normal. Enlargement of solitary glands in small intestine. Thyroid enlarged. Body jaundiced.

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CASE 3.—Emily R., æt. 23, single, admitted into Mary, September 16th, 1889, under Dr. Pitt. (Pye-Smith, 1889, Report 107). Previous illnesses and family history unimportant. In March last noticed weakness and vomited after food. In April throat was found to be enlarged. For one month before admission has vomited every morning. Bowels constipated. Has been very nervous since beginning of illness.

*Condition on admission.*—Nipples and axillæ pigmented. Temperature 100°, respiration quick and hurried. Pulse 180. Wriggles about in bed and utters short cries. Tongue furred. Urine normal. Enlarged thyroid, exophthalmos marked. She came up from Wales the day before admission and was taken with violent vomiting on the next day. September 17th, temperature 100°, pulse and respiration not noted. Has been vomiting. In the morning was cyanosed. Injection of morphia given in the evening and she died 9.45 a.m. next morning. Report very meagre.

*At the autopsy.*—Thymus much enlarged. Liver fatty, stomach and intestines congested. Kidneys tough (9 ounces). Brain normal, except for recent hæmorrhage between the optic thalamus and the caudate nucleus, which Dr. Pitt thought of no importance.

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CASE 4.—Mary J., æt. 23, single, admitted into Mary under Dr. Perry, August 21st, 1889 (Taylor, 1889, Report 122). Died August 25th, 1889. Family history unimportant. Symptoms commenced two years ago.

*Condition on admission.*—Temperature 99°, pulse 130, respiration 60. Is very excited and difficult to obtain information from. Signs of Graves' disease well marked. Bowels regular. She had travelled from Wales on day of admission, and had apparently taken a good deal of spirits on the journey. She had to be put under restraint the first night in hospital. August 24th, nervous condition unchanged, temperature 98.8°, pulse 138, respiration 39. August 25th, patient very delirious, breathing very rapid. Chloroform administered. Later feeding with nasal tube. She got gradually worse and died at 8 p.m.

*Autopsy* limited to heart, which was found healthy.

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CASE 5.—Martha B., æt. 30, single, admitted into Mary under Dr. Hale White, July 25th, 1894. Died August 8th, 1894. (Hale White, 1894, Report 258.) Personal and family history unimportant. First noticed the present disease two years ago. No abdominal pain; bowels normal.

*Condition on admission.*—Temperature 98°, pulse 116, signs of Graves' disease well marked. Is well nourished and healthy looking. Rather hysterical. On August 7th Mr. Dunn removed the right lobe of the thyroid. She took the anæsthetic well, and on coming round the pulse and respiration were as before the operation. August 8th. She did not pass a good night. This morning temperature 101·6°, pulse 160, respiration 56. Is somewhat cyanosed and sweating profusely. Tongue dry and furred. At 1 p.m. temperature 103°, pulse 172, respiration 68. Is "off her head." Bowels opened. Brandy injected and oxygen administered. Died at 4.15 p.m.

*At the autopsy.*—Two and a half ounces of thyroid gland left. Brain not examined. Eyes still prominent. Other organs normal.

**CASE 6.**—Jane C., æt. 22, single, admitted August 3rd, 1896, into Mary under Dr. Taylor. Died August 7th, 1896 (Taylor, 1896, Report 216.). History of heart disease in the family. Nine months ago was told by a doctor she had Graves' disease. For about fourteen days before admission she has had diarrhœa and vomiting. Says she is very nervous. On July 30th she noticed pains in the left wrist and later in the shoulder and ankle.

*Condition on admission.*—Is flushed and sweating. Temperature 100°, pulse 100, respiration 24. There appears to be the same slight swelling, but no redness of the left wrist. She complains of pain in the left ankle as well, but this looks normal. Respiratory, cardiac and urinary systems normal. Thyroid enlarged. Eyes not prominent. August 5th. Is not so well. Is restless and complains of pain in the legs and between the shoulders. Joints not painful. Temperature 101°, pulse 136, respiration 30. August 7th. Râles noticed at the left base. Temperature 104°, pulse 152, respiration 44. Muttering delirium, attempts to get out of bed. Dr. Perry saw patient and did not consider she had pneumonia, but that her condition was due to the disease of the thyroid. At 4 p.m. temperature 105, pulse 152, respiration 44, full and deep. She died in a convulsion whilst being sponged. Diarrhœa persisted all the time she was in hospital.

*Autopsy.*—Brain not examined. Prominence of the eyes had disappeared. Thymus persistent, and as large as the fully developed gland in the child. Thyroid enlarged and its isthmus contained a cyst. No laryngeal obstruction. and no evidence of pressure on the trachea. Peyer's patches and solitary follicles prominent. Other organs normal, except for congestion of left lung.

**CASE 7.**—Ellen P., æt. 36, married. Admitted on September 7th, 1896, into Mary, under Dr. Taylor. Died October 1st, 1896 (Taylor, 1896, Report 253). *Family history*, unimportant. *Personal history*—Present disease, started at age of 17. Four years ago she had a miscarriage (no mention of any children being born). Married at the age of 28. In October last had pneumonia, pleurisy and nephritis, with delirium. Has had rheumatism. Says she has wasted considerably.

*Condition on admission.*—Symptoms of Graves' disease, well marked. Legs cedematous, and some pigmentation of face, arms and legs. Systolic apical bruit traceable to axilla. Temperature 99·4°, pulse 120, respiration 28. Urine contains albumen (13 parts per 1,000). Other systems normal. Digitalis and belladonna prescribed. September 9th, patient has had diarrhœa in the night. Temperature 100·4°, pulse 120. September 16th, diarrhœa has continued.

Pulse 130 and irregular. Patient perspires profusely in her sleep. Temperature 100·4. September 18th, complains of sharp pain under left scapula, was delirious at night, getting out of bed. Pulse, feeble, 150. September 22nd, temperature 103°, pulse 120, respiration 36. Has continued delirious. September 24th, tongue dry and tremulous. Condition unchanged. There is ulceration of left cornea. Patient continued to have an irregular temperature, sometimes reaching 101°, with respiration of 40–50, and a weak, irregular pulse, varying between 80 and 140. Delirium continued until her death, on October 1st.

*Autopsy*.—Brain not examined. Small portion of thymus persisting. Thyroid enlarged in both lobes. Numerous hæmorrhages beneath the pleuræ all over the lungs. Lungs œdematous, but not pneumonic. Trachea not pressed on, nor deformed. Heart, eleven ounces, slightly enlarged. Mucous membrane of intestinal tract reddened throughout. Follicles not prominent. Kidneys, twenty-three ounces, but appeared quite normal in structure.

CASE 8.—Sarah B., æt. 37, admitted under Dr. Perry into Miriam, June 18th, 1897. Died June 25th, 1897 (Clinical, 1897, Report 282). She is married, has had no children, but had a miscarriage three years ago. Had acute rheumatism in September, 1896, and congestion of the lungs in the following December. Present illness dates from acute rheumatism, commencing with weakness and palpitation. Family history unimportant.

*Condition on admission*.—Signs of Graves' disease well marked. Talks constantly and cannot keep still, speech blurred. Pulse 108, temperature 98·4°. A trace of albumen in urine. Other systems normal. June 19th. Dr. Perry thought she had an old mitral endocarditis. Leiter's tube ordered to neck. Strophanthus bromides, arsenic, belladonna internally. Breathing rapid and catchy (36). Dr. Perry said the prognosis was bad on this account. June 23rd, patient very drowsy. Temperature 99°, pulse 148, respiration 44. She becomes very blue at intervals. June 24th, was continually getting out of bed in the night. Temperature 101°, pulse 152, respiration 44. Is semi-comatose and of a very livid colour. June 25th, condition remained the same till death at 1.15 a.m. The heart stopped beating before respiration ceased.

*At the autopsy*.—Thymus present, weighing one and a half ounces. Thyroid enlarged. Heart, eleven and a half ounces; valves normal. Kidneys congested, fifteen ounces. Stomach and intestines congested, and the latter contained blood. Solitary follicles of small intestine prominent. Trachea not at all compressed. Brain, lungs, liver, etc., normal.

CASE 9.—Lily W., æt. 32, single, admitted into Mary under Dr. Taylor, August 12th, 1896. Died October 10th, 1896 (Taylor, 1896, Report 232). Was admitted into Miriam on December 9th, 1891, for Graves' disease, the symptoms of which had appeared in the preceding year. She was in hospital five weeks and was discharged improved. Since discharge she has done her work at intervals, but has suffered much from palpitation.

*Condition on admission*.—Pulse 100, temperature 98°, respiration 30. Exophthalmos, enlarged thyroid, and tremors of hands present; is thin. Systolic murmur over pulmonary and mitral areas. Urine normal. Mental condition not mentioned. August 28th, pulse 86, temperature 98°, August 31st, very restless at night; palpitation still troublesome. September 16th, has been

much troubled with vomiting for last few days, pulse 110, respiration 40. Has a great deal of pain and tenderness over upper part of abdomen. September 26th, has been much better for the last three days, pulse 100. September 29th, erythematous rash over legs. Is worse, pulse 112. October 2nd, complains of headache, is very despondent, erythema still present. October 6th, was better yesterday, but to-day vomiting has recurred and she is worse. October 7th, much exhausted, respiration 60, pulse very rapid, mental faculties deranged. October 10th, tossing about and crying hysterically, temperature last night 99°, to-day 101°, respiration 32, pulse 160, bowels open five times. She died at midnight.

*Autopsy.*—Brain 46 ounces. Branches of middle cerebral artery which supplied operculum and part of cerebrum just anterior to it, shew ante-mortem thrombosis. Acute softening had occurred in this area. Two small spots of softening in the front of the lower third of the pons on both sides. Eyeballs prominent. Thyroid much enlarged. No compression of trachea. All other organs quite normal.

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CASE 10.—Maud H., æt. 24, single, admitted into Mary under Dr. Hale White, February 27th, 1900. Died April 6th, 1900 (Hale White, 1900, Report 94). Admitted for Graves' disease. Family history good. Previous diseases unimportant. Influenza a year ago, after which she noticed breathlessness and palpitation. In November was told the thyroid was enlarged, and she noticed prominence of eyes. Menstruation irregular and scanty.

*Condition on admission.*—Pulse 132, temperature 99°. Is flushed and rather nervous. Urine normal. Tremors present. Prescribed Belladonna, Digitalis, and Bromide. March 5th, she has vomited all day. March 6th, has vomited during the night; had a fit of crying this morning which lasted some time. Temperature 99°, pulse 120, respiration 36. March 8th, better. Vomiting has continued since March 4th, but has now stopped. March 15th, she has dermatitis on the arm and ear. Skin is red and itches. Pulse 96, respiration 28, temperature 98°. March 19th, is worse; complains of pain all over. Dermatitis continues. Temperature 101·2°, pulse 132. March 21st, throat swollen and red. March 27th, is much worse. Temperature 102°, pulse 160, respiration 40. She has lost about twenty pounds in weight since admission. She has been delirious off and on for the last week; has a good deal of bronchitis. March 31st, condition unaltered. Dermatitis and sickness continue. Temperature 101°, pulse 160, respiration 50. April 4th, she takes practically no nourishment. Temperature 101°, pulse 160, respiration 80. She has diarrhoea. Lobar pneumonia diagnosed. Delirium has continued. April 6th, she died at 6 a.m.

No autopsy. This patient was not examined often by request, hence the report is meagre.

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CASE 11.—Ellen G., æt. 26, single, admitted June 2nd, 1900, into Mary, under Dr. Hale White. Died June 29th, 1900 (Hale White, 1900, Report 192). *Family history.*—One sister has palpitation and enlarged thyroid. *Personal history.*—Influenza and congestion of the lungs last Christmas. *Present illness.*—In July, 1898, noticed tremors and headache. In the following October, throat was found to be enlarged. Says she has lost weight, and that the skin of her face has become darker.

*Condition on admission.*—Pulse 126, temperature 98·4°, respiration 34. Signs of Graves' disease, well marked. Mental condition stable. Some albuminuria. Other systems normal. Ordered digitalis, bromide and belladonna. She progressed well till June 18th, when temperature rose to 100·4°, pulse 128, and she had some tonsillitis. June 23rd, is much better. Throat well. Temperature has remained between 98° and 101°, pulse 100 to 120, respiration 30 to 40. Has some diarrhoea. June 26th, patient very depressed and melancholic in appearance. Temperature remains up. June 28th, temperature 103°, pulse 140, respiration 40. Has had a very bad night, delirious, getting out of bed and refusing food. To day is listless, eyes glazed and fixed, limbs cold and clammy. June 29th, condition unaltered, delirium continues. Oxygen was given. She seems to have a collection of mucus in her throat. She died at 8.15 p.m.

*Autopsy.*—Brain not examined. Traces of thymus present. Thyroid enlarged, and contained several small cysts. Some bulging inwards of the wall of the trachea opposite the fourth, fifth and sixth rings. Just below the cricoid the trachea was also narrower than usual. Other organs normal.

CASE 12.—Catherine W., æt. 25, admitted May 13th, 1899, into Dorcas, under Mr. Jacobson. Died May 17th, 1899. Comes in for pulsating tumour of thyroid and exophthalmos. Married. Family history unimportant. Had typhoid three and a half years ago, and a miscarriage eighteen months ago, after which tumour first noticed. Has caused no pain, but it has increased. Has experienced choking fits from time to time. Is easily agitated and nervous, and cries frequently.

*Condition on admission.*—Thyroid enlarged, bowels normal, appetite good. Temperature 100°, pulse 128, respiration 32. May 16th. Operation 4 p.m. She took the anæsthetic well, and the right lobe of the thyroid was removed. At 10 p.m. temperature 103°, pulse 140, respiration 32. Restless, talkative, throws her arms about. Pulse increasing and feeble, is delirious and movements seem quite uncontrolled. Died at 4.15 p.m. May 17th.

*Autopsy.*—Brain not examined. Thymus large. Wound not septic. Thyroid substance very pale. Right lobe had been removed. Heart ten ounces, normal. Liver very pale, ? fatty. Other organs normal.

CASE 13.—Harriet H., æt. 48, admitted under Mr. Jacobson into Dorcas March 9th, 1900. Died March 12th, 1900. Came in for swelling in neck. Is married, has had six children all healthy. Twelve months ago swelling noticed, it gradually increased and she began to lose weight rapidly. Went to several places for change and rest. As a result weight increased. On return home she gradually got worse.

*Condition on admission.*—Looks in bad health and seems of a nervous temperament, is wasted and eyes protrude. Thyroid enlarged. No dyspnoea. Von Graefe's sign present. Feels quite well except for weakness and the swelling. Pulse 132, temperature 98°, respiration 20. March 12th, delirious, gets out of bed, temperature, 101°, flushed and sweating, pulse 144. Condition thought by husband due to shock at seeing a patient taken to the operating theatre. She got worse and died.

*Autopsy.*—Thyroid enlarged. Thymus persistent. No evidence of pressure on trachea. Old healed gastric ulcer causing some deformity of pyloric end of stomach, but no stenosis. Brain and other viscera normal.





# ACUTE INTESTINAL OBSTRUCTION CAUSED BY THE ILEUM BECOMING ADHERENT TO A LITHOPEDION.

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By J. H. BRYANT, M.D.

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I HAVE thought this case worthy of publication for two reasons, firstly on account of the extreme rarity of the occurrence of a lithopedion, and secondly because, as far as I know, acute intestinal obstruction from this cause is unique.

A very full account of lithopedions is given in the Bulletin of the Johns Hopkins Hospital, vol. viii., No. 80, p. 221, by Dr. J. G. Clark. He reports an interesting example of this condition, and gives a review of the cases hitherto published. He refers to Kuchenmeister's table of forty-seven cases reported between the years 1582 and 1880, and mentions eighteen others published subsequently, in addition to his own case, in all, a list of sixty-seven. No mention is made of any of these cases terminating fatally as a result of acute intestinal obstruction from the intestine becoming adherent to the lithopedion.

In the case recorded below the cause of the intestinal obstruction was, partly kinking and partly strangulation. A small loop of

ileum was found to be adherent to the body of the lithopedion in two places; through the small aperture thus formed an adjacent coil of ileum had passed and become strangulated. There was also considerable kinking of the ileum at the two points where it was adherent to the body of the lithopedion, and there was another piece of ileum higher up which was also kinked, on account of its being adherent to the head of the lithopedion. The kinking alone was not sufficient to account for the acute symptoms, which must have been caused by the loop of the ileum becoming strangulated between the adherent loop and the body of the lithopedion.

The presence of the lithopedion was not suspected during life. The only possible indication of it was the history of five months' amenorrhœa, which had occurred two and a half years before. There was no previous evidence to suggest or indicate in any way a ruptured tubal pregnancy. The hard mass which was felt before and at the time of the operation was considered to be a malignant growth. It was unfortunate that the patient's condition precluded the possibility of investigating the tumour at the time of the operation, as from the post-mortem examination, I should say, it would have been quite possible to have dissected out the lithopedion and to have completely removed the obstruction. I am indebted to Dr. Perry for permission to publish the clinical notes and to Dr. Stevens for his excellent drawing.

Hannah H., 37, was admitted under the care of Dr. Perry, on November 28th, 1899, for intestinal obstruction (clinical clerk, E. Cohen). About two and a half years ago she had an attack of intestinal obstruction which lasted about five days. The bowels were eventually relieved by enemata. Since then she had never had any trouble with her bowels and had been quite well. On Thursday, November 16th, she partook of a big supper, and after going to bed complained of a pain in her abdomen. On the next day she was able to get up and do her work. The bowels were opened on the 17th, but the pain soon afterwards came on again, and as it continued, a doctor was called in.

Numerous efforts were made to relieve the bowels by means of purgatives and enemata, but without effect. Mr. Dunn was asked to see the patient on the 27th, and he advised her removal to the hospital with a view to laparotomy in order to find the cause of the obstruction and if possible to remove it. On November 23rd, she had a bad attack of vomiting which lasted about twenty-four hours. The vomit had a very foul odour and she stated that it smelt like a motion. She did not vomit on the 26th. Two and a half years ago she gave a history of five months' amenorrhœa; before and after the menstrual disturbance she had always been regular. There was no history of pain or anæmia, and, as far as she knew, she had never been pregnant. There was no history of any previous pelvic trouble.

*Condition on admission.*—Temperature 99·2°, respiration 24, pulse 104. She was rather collapsed. Her tongue was dry and furred. Her eyes were sunken and she had an anxious expression. She did not appear to be in pain, and was not wasted or cachectic looking. There was no darkening of the areolæ, and there were no lineæ striæ on the mammæ. She complained of a constant desire to pass her urine. The abdomen was distended. On palpation, a hard mass could be felt in the lower part of the abdomen but extending almost as high as the umbilicus in the median line. On the right side it appeared to extend a little higher than the umbilicus, and it was fairly well defined and was movable. On percussion, a tympanitic note could be obtained all over the abdomen except in the right iliac fossa, where there was dulness. The outline of a distended coil of intestine could be seen crossing the abdomen just above the umbilicus. No peristalsis was visible. The heart and lungs appeared to be normal. The urine was 1020; there was no albumen, sugar or blood present.

November 28th. She was ordered a milk diet and appeared very comfortable in bed. At 1 a.m. she became restless and was given an injection of Morphia gr.  $\frac{1}{8}$ , and Atropine Sulphate gr.  $\frac{1}{16}$ . After this she slept for a time and was comfortable.

On November 29th she looked worse, and her eyes were sunken. Pulse 108, respiration 25, temperature 96·2°. A vaginal examination was made and the cervix was found to be anteverted. On bimanual examination, a large, undefined, hard mass could be made out in the hypogastric and lower umbilical region, which appeared to be fixed to the uterus. Dr. Perry and Mr. Dunn saw her and an exploratory operation was decided on in order to determine, and if possible to remove, the cause of the obstruction. Mr. Dunn made an incision in the median-line of the abdomen about five inches in length and commencing three inches above the umbilicus. On opening the peritoneal cavity the transverse colon was found to be collapsed. He explored the abdomen with the hand and found a very hard stony-like mass just above the pubes. It was considered to be a growth binding down and kinking some coils of small intestine. Owing to the extremely serious condition of the patient it was thought inadvisable to attempt to remove the hard mass, and so a portion of the small intestine, which was found to be very distended, was pulled out, and a Paul's tube was inserted as near to the obstruction as possible. The abdomen was then closed, a small portion of the intestine containing the Paul's tube being brought out of the lower part of the wound.

She took the anæsthetic badly, and was very collapsed afterwards. She did not rally after the operation, and gradually sank and died at 4 a.m.

I performed the necropsy ten hours after death. There were no signs of decomposition. Rigor mortis was well marked. There was an incision about five inches in length in the median line of the abdomen commencing three inches above the umbilicus, the upper portion of which had been united by gut sutures. A piece of small intestine in which was situated a Paul's tube occupied the lower part of the incision. The lungs and pleuræ were normal. The heart weighed 234 grammes, and was normal in appearance. The mouth, pharynx, œsophagus and stomach were normal. On opening the peritoneal cavity the hard mass felt during the operation proved to be a lithopedion; it was situated in the median

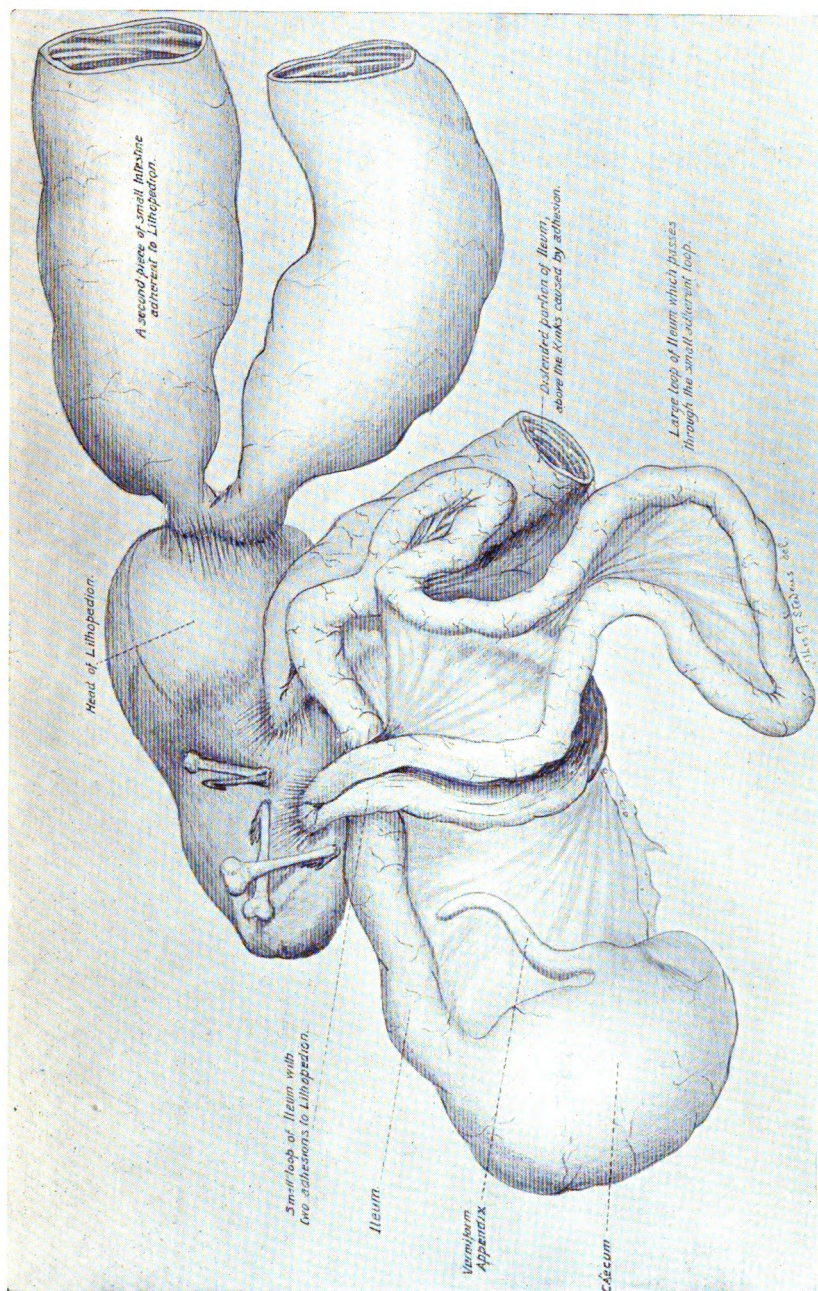
line between the pubes and umbilicus, reaching to the level of the latter. The head was pointing to the left. It was found to be attached to several coils of small intestine, and also by a few fibrous adhesions to the uterus and right broad ligament. The jejunum had been opened about one and a half metres from the duodenum. A coil of the lower end of the ileum measuring about 60 centimetres was completely collapsed, it being strangulated by having passed through a small aperture which was formed by a small portion of the ileum immediately above having become adherent to the body of the lithopedion in two places close together. At both points where the gut was adherent it was sharply kinked, so that the obstruction was partly due to kinking and partly to strangulation. A little higher up another piece of the ileum was adherent to the head of the lithopedion and this was also kinked but not so markedly as the loop below. The uterus measured 7·5 centimetres in length. The cervix was nulliparous. There were general pelvic adhesions. Both Fallopian tubes were found to be running backwards over the surface of the ovaries and were adherent in Douglas' pouch. The left ovary was normal in size and appearance. Attached to the right ovary was a spherical tumour measuring 5·5 centimetres in diameter; it was firmly fixed to adjacent parts by firm fibrous adhesions and it was with difficulty freed from these attachments. On section it was reddish brown and appeared to be made up principally of altered blood. It appeared to be the remains of the old placenta. Dr. Stevens very kindly cut some sections and found degenerated chorionic villi and said the tumour was undoubtedly made up of placental tissue with blood clot. The lithopedion was found to be lying almost free in the peritoneal cavity; there were a few adhesions attaching it to the uterus and right broad ligament. It was in a condition of general flexion as if it had been subjected to much pressure. The head was flexed on the thorax and there was marked kyphosis. The feet, legs, hands and arms were fully flexed. The left knee was tucked under the middle of the right femur, and the right leg was lying across the middle of the

left leg. The arms were placed close to the sides of the thorax. The head was flattened from side to side. The measurements were :—

<i>Head</i> —Biparietal diameter	...	...	5·1 cms.
Vertical	...	...	5·5 cms.
Antero-posterior	...	...	6·4 cms.
Circumference	...	...	19 cms.
The length of the Body was	...	...	7·5 cms.
„ Femur „	...	...	4·5 cms.
„ Tibia „	...	...	4·2 cms.
„ Radius and Ulna was	...	...	3·5 cms.
„ Humerus „	...	...	4 cms.

The measurements of the lithopedion very nearly corresponded to those of a five months' fœtus.

*Acute Intestinal Obstruction, caused by the Ileum becoming adherent to a Lithopedion.*



Acute intestinal obstruction, caused by the Ileum becoming adherent to a Lithopedion. T. G. STEVENS (del.)





# ON THE RADICAL OPERATIONS FOR UTERINE CANCER IN GUY'S HOSPITAL FROM 1886 TO 1899.

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By THOS. G. STEVENS, M.D.

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THE cases which form the basis of this paper were treated in Guy's Hospital between the years 1886 and 1899, and the series includes all those cases of cancer of the uterus which were submitted to radical operations. By radical operations it is intended to mean, deliberately planned cutting operations, by which, as far as the naked eye could see, all the growth was removed with a part or the whole of the uterus. No cases have been included in which the growth was removed by the curette or *écraseur*, with subsequent treatment by cauterising agents. No doubt, many good results were obtained by these methods previous to 1886, but at the same time the accidents attending such operations were numerous, and owing to the impossibility of gauging the exact amount of growth removed, or the exact depth to which a caustic would burn, the operator never could be sure whether the whole of the macroscopic growth had been removed. So practically the only operations included in this series are :—Supra-vaginal amputations of the cervix uteri, and cases of total vaginal hysterectomy, with a few cases in which the abdomen had to be opened from above, owing to some unusual

difficulty in completing the operation from the vagina. These operations can be fairly claimed to be radical, because they aim at deliberately cutting out the growth, with a part or the whole of the uterus. Of late years the term "radical" has been applied to such sweeping operations as opening the abdomen and removing not only the uterus and growth, but also all the fat and glands in the pelvis likely to be at the time or later infected with secondary growth. Such an operation, however, can no more claim to be really radical than a simple hysterectomy, because if a cancer of the uterus has already infected the glands in the pelvis it is by all canons of surgery already too far advanced for any operation to be undertaken with even a remote prospect of cure. The pelvic glands, fat and connective tissue cannot at present be placed in the same category as the similar structures in the axilla, for the glands in the pelvis cannot yet be removed so easily and safely, as the axilla can be cleared out for mammary cancer. Fortunately glandular infection in cases of uterine cancer comes late in the progress of the case, and so has very little influence on the feasibility of any operation being performed. Fixation of the uterus by infiltration of the surrounding connective tissue structures will give an indication of the uselessness of operative procedures long before glands become obviously infected.

The cases included in this paper cannot on the whole be considered favourable for operative measures. They mostly presented rather long histories, many had large growths, and in some the growth had already invaded the vaginal walls and surrounding connective tissue to some extent, and yet these were the most favourable cases which could be picked out from all those admitted to the gynæcological ward in the fourteen years from 1886 to 1899 inclusive. Altogether 274 cases of uterine cancer were admitted to the Hospital during that period, and of these, only 102, or 37·2 per cent., were deemed at all favourable for any radical operation. Of the other 172 cases, most, on careful examination, were found to be too far advanced for any operation to give the remotest chance of even relief, not to speak of cure. Operations were attempted in a few instances only to be abandoned, because it was found that the operation

could not be completed owing to infiltration of surrounding parts, such as the bladder or broad ligaments. In some of these cases the bladder was accidentally opened during the stripping of that viscus from the uterus, owing to the growth having already spread through its wall. As many of these cases were received direct into the ward from practitioners in the provinces, these figures cannot be used to show what percentage of the cases of uterine cancer presenting themselves for the first time in the out-patient department were favourable for operation. There is no doubt, however, that the percentage of cases of uterine cancer fit for removal seen in the out-patient department is much smaller than 37·2 per cent. This only serves to emphasize what has already been often said by writers on the subject, that cases of uterine cancer nearly always seek advice when it is already too late for any operative procedure to be undertaken.

*Previous history of these 102 cases.*—The average duration of the symptoms complained of before admission to the Hospital was seven and a half months. There was some difficulty in arriving at the real duration of the history in some of these cases, owing to the probable presence of antecedent uterine disease, such as endometritis, cervical erosions, etc., and some discretion had to be exercised in judging when the symptoms of the growth really began. The duration varied very much, the shortest time mentioned being one month and the longest two years. Altogether there were 26 cases in which the duration of symptoms was twelve months and upwards. Nevertheless, from an analysis of these 26 cases it is clear that the long history did prejudice the final results. Four died from the immediate effects of the operation, or 15·36 per cent., whereas of the remaining 76 cases nine died, or 11 per cent. Four are alive now in 1901, or 15·36 per cent., whilst of the others thirteen are alive, or 17·1 per cent. Although, from these small figures, it would not be justifiable to draw any positive conclusions, we may, however, infer that the longer the duration of the history the less likely is the result to be favourable in the end.

*Etiology.*—Very few conclusions can be drawn from a consideration of these cases with regard to their etiology. Ninety-

nine occurred in married women, of whom seven had had no children. Three only occurred in single women. The cervix was the part affected in ninety-four married, and in two single women. The body of the uterus was affected in five married and only in one single woman. This gives the proportion of cancers of the body of the uterus to those of the cervix at six to ninety-six, or 5·8 per cent. This perhaps is rather below the average, although according to Schroeder's figures it is rather less than 2 per cent. In a considerable number of cases it was stated in the reports that old tears of the cervix were present. Many cases gave histories of long standing uterine discharges, showing that they had had endometritis or cervical catarrhs for long periods previous to the onset of the malignant growth. In five cases the cancer occurred in a prolapsed uterus of some years' duration. In one case the disease deciduoma malignum followed on hydatidiform degeneration of the chorion. These few facts bear out the common statements, that cancer of the uterus is common in married women who have borne children, is often associated with old lacerations of the uterus, and probably that pre-existing cervical catarrh or corporeal endometritis stands in some direct causal relation to the disease.

As to the ages of the patients, the youngest was 26 and the oldest was 66. The average age of all the cases was 45. There were four patients under 30, one of 26, two of 27, and one of 29; twelve patients under 35. So that while no doubt the great majority of cases are between 40 and 50 years of age, it must not be lost sight of that cancer of the uterus is by no means uncommon under 30, and fairly common under 35.

*Symptoms complained of.*—The commonest symptom complained of, was irregular bleeding, not associated with menstruation. The bleeding varied from a slight intermenstrual sanious discharge to veritable "floodings." Often the bleeding had no relation to physical exertion or other cause, but in a good proportion of cases it first occurred at coitus. Particularly in cases past the menopause, bleeding was the first symptom noted. Most of the patients had complained of discharges for varying periods, but only in a very few instances was it distinctly stated

that an early symptom was a *watery* discharge. A watery discharge is often stated by writers to be the first symptom of cancer of the uterus, but in this series it was not so. The watery discharges mentioned in this series commonly continued after the first hæmorrhage had occurred. Offensiveness of the discharges is mentioned as a very common sign, but is not really very important, because it merely indicates as a rule the presence of early ulceration of the growth. Scarcely any of the cases are noted to have the horrible stinking discharge of long standing cancers.

*Pain.*—Forty-five of the cases had no pain, or there was no mention of pain in their reports. Of the others it is difficult to judge whether the pain mentioned was really connected with the growth, or was a part of some old uterine congestion, backache being often the only pain complained of. Unless pain is of the typical radiating character of cancer it cannot be distinctly stated to be a symptom of the disease. Considered as critically as this, there are very few cases in the series in which pain is at all a prominent symptom. For instance, in some of the cases with long histories (Case 51 for instance, with eighteen months' history), it is distinctly stated that there was *no pain*, and others show the same point. This serves to demonstrate the oft-repeated fact, that pain is a late symptom of uterine cancer.

*Wasting*, certainly is not an early symptom of uterine cancer; only 36 cases of the series are stated to have lost flesh before admission to the Hospital. Many of the patients are mentioned as having looked fat and healthy, and appeared to be the least likely subjects to have malignant disease.

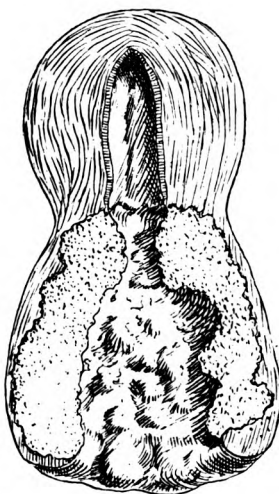
*Micturition.*—Twenty-four cases are stated to have troubles connected with the bladder or urethra. The common symptoms were painful and frequent micturition, probably due to slight urethritis caused by discharges which infected the urethra. Occasionally, no doubt, micturition troubles came on as a result of the growth encroaching on the bladder wall, where it is in contact with the uterus. For instance, in Cases 45, 54, 74 and 76 the patients had micturition troubles, and it is stated that the bladder stripped from the uterus with difficulty, no doubt owing



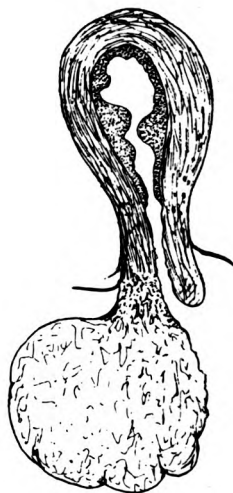
CASE 76.—A little more than half natural size. Antero-posterior section. Shows the growth and the great elongation of the supra-vaginal cervix.



CASE 82.—A little more than half natural size. Antero-posterior section. Shows the growth involving the posterior lip, and posterior vaginal wall.

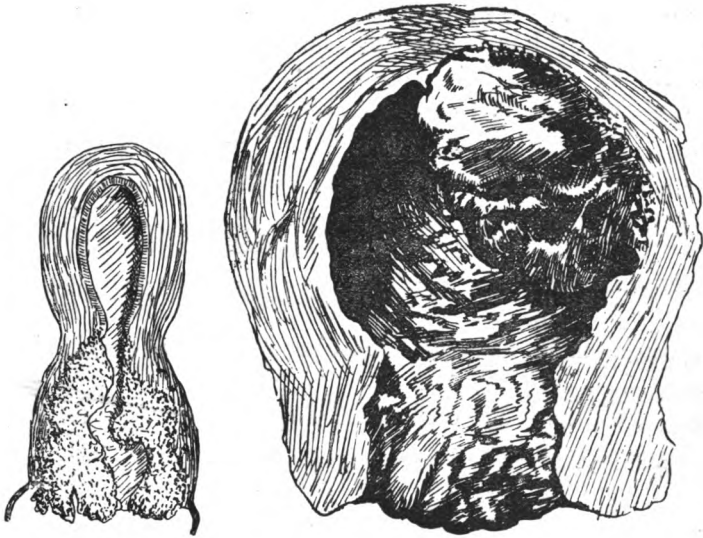


CASE 80.—Half natural size. Antero-posterior section of uterus. The dotted area shows the limits of the growth.



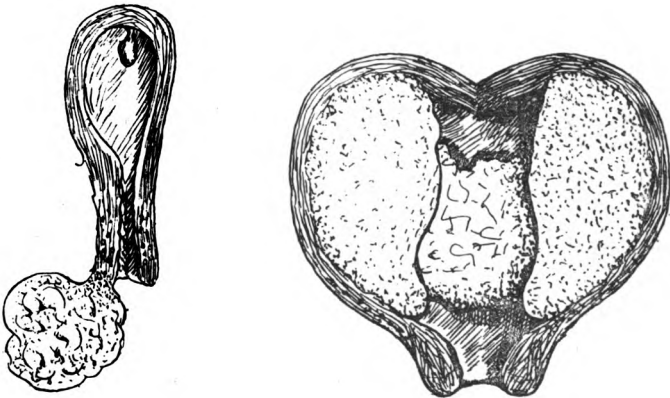
CASE 88.—About half natural size. Antero-posterior section of uterus, showing adenomatous endometrium and growth projecting from the anterior lip of the external os.

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CASE 94.—About half natural size. The dotted area shows the limits of the growth.

CASE 84.—Uterus split open posteriorly. Two-thirds natural size. Shows the growth projecting into the uterine cavity, and the thickened subinvoluting uterine wall.



CASE 100.—About half natural size. Antero-posterior section of uterus. Shows the projecting growth, and a small adenomatous polypus at the fundus.

CASE 102.—Uterus split open posteriorly. Two-thirds natural size. The dotted area shows the extent of the growth.

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to the close proximity of the growth. The micturition troubles may be caused in two ways; either by the mechanical interference of the growth, causing pain and frequent micturition owing to the inability of the bladder to distend properly, or to a slight catarrhal cystitis over the part of the bladder close to the growth.

*Defæcation* troubles were very rare in the series; only in a very few was there any mention of pain on defæcation.

*Pathological anatomy.*—The kind of growth in each individual case cannot always be determined with certainty from the reports, because in the earlier cases no microscopical examinations were made. Of the 102 cases, as far as can be ascertained, there were eighty-five of glandular carcinoma of the cervix, eight of squamous epithelioma, three of sarcoma, five of carcinoma of the body of the uterus, and one of deciduoma malignum. These figures, however, must be viewed somewhat sceptically, because in all probability the percentage of cases of squamous epithelioma was really greater. There were roughly five types of growth classified according to their naked-eye characters.

1. Growths of the cervix presenting shallow ulcers with hard everted edges, generally of slow growth and probably commencing on the vaginal aspect of the cervix. The drawing of Case 82 is of this type of growth.

2. Mushroom-shaped growths, which, commencing first inside the external os, gradually expand the lower portion of the cervix and fungating into the vagina produce a flat expansion not unlike a mushroom. These were usually soft growths of the glandular carcinoma type, and formed the great majority of the cases. The drawings of Cases 76 and 94 seem to be of this type.

3. Projecting solid almost pedunculated growths. These apparently had the same origin as variety No. 2, but as they had not ulcerated on the surface, produced a more or less smooth projecting mass. The drawings of Cases 88 and 100 were of this variety.

4. Cauliflower growths; soft, very friable lobulated growths attached by a small base and proliferating into the vagina. These probably have their origin in the columnar epithelium



lining the cervix, but occasionally they may take origin from the squamous vaginal epithelium, chiefly the rete Malpighii layer.

5. Growths which commenced a little way up the cervical canal, and spreading laterally widen the cervix and appear later at the external os. The drawing of Case 80 seems to be one of this type.

The growths of the body of the uterus were generally rough and irregular on the surface from breaking down of the superficial layers. These naturally are only of one type, glandular carcinoma commencing from the columnar epithelium lining the body of the uterus. The drawing of Case 102 shows the very extensive growth infiltrating the uterine wall almost everywhere. The only case of deciduoma malignum was absolutely typical. The drawing of Case 84 shows the appearance of the somewhat uneven-surfaced growth projecting into the uterine cavity and infiltrating the wall. Microscopically it consisted of the usual two elements, namely, large epithelial looking cells with round or oval nuclei, and masses of giant cells, the syncytium, here and there filling up interstices between the masses of the other cells. This growth is generally held to be a sarcoma, but as the subject is very controversial no definite statement is necessary here.

The *diagnosis* of these cases, especially those of cervical cancer, usually presented no difficulty. The appearance of the growths as described above, the history of the case and the ready bleeding on examination, generally left not the slightest doubt as to the nature of the disease. This, too, is usually the case in hospital practice, where patients commonly seek advice after the symptoms have existed some time. However, in cases where the disease of the cervix is not self evident, and almost always in cases of disease of the body of the uterus, it is necessary to make sections of curetted pieces for microscopical examination before a definite diagnosis can be arrived at. In many of the latter cases of this series, pieces of growth were removed for microscopical examination and proved of the greatest value in diagnosis. The value of the microscope as an aid to diagnosis in these cases is often

called in question, and the reason is not far to seek. Too often pieces of growth are removed by the curette and sent to a pathologist in some indifferent solution, such as carbolic lotion or weak perchloride of mercury, or even half dried and wrapped in indiarubber tissue. From such specimens only very indifferent sections can be made, and far from being useful in diagnosis, they become a stumbling block. Also properly prepared specimens, if badly cut, *i.e.*, too thick, and improperly stained, are worse than useless as an aid to diagnosis. To be useful the curetted specimens must be *fixed* in some solution such as saturated solution of corrosive sublimate, absolute alcohol or saturated solution of picric acid in water. The best possible sections must be then cut by the paraffin method, for no other method gives such perfect results. Sections prepared in this way will always give useful information to a skilled histologist. The specimens will naturally fall into three categories, those which are undoubtedly cancer, those which as certainly are not, and those which are doubtful. The latter cases will consist of adenomata and cervical erosions, which may or may not be becoming malignant. Such cases must of necessity always leave a doubt in the mind of the histologist. But the very fact of a specimen having been made, and a doubt as to its identity being present, will be of the very greatest importance to the patient and the surgeon, because it will ensure that particular case being carefully watched, and immediate operative steps undertaken if the symptoms seem at all progressive. The conclusion to be drawn from this is, that to make the microscope useful as an aid to diagnosis, the specimens must be most carefully prepared, the sections the best possible, and the histologist selected for their examination a skilled one.

*Treatment.* — Supra-vaginal amputation of the cervix was performed thirty-six times. The procedure was the same in all cases, with but slight modifications. The patient was placed in the lithotomy position, the legs being held by a Clover's crutch. Then all hair was shaved from the vulva and the external genitals and the vagina thoroughly cleansed. A Symon's or other speculum was then inserted into the vagina and the

cervix or growth seized with tenacula and drawn down to the vulval outlet. After ascertaining the position of the lowest part of the bladder on the cervix by means of a bladder sound, an incision was made as wide of the growth as possible from side to side on the anterior surface of the cervix. The bladder was then separated from the uterus by the finger or blunt dissector, the separation being carried as high up in front as it was deemed necessary to amputate the cervix. Next the cervix was pulled forwards and upwards and an incision made through the vaginal wall posteriorly, with its ends joining those of the anterior cut. In the earlier operations care was taken not to open Douglas' pouch at this stage, but to strip up the peritoneum off the posterior surface of the uterus. In the later cases the peritoneum was fearlessly opened because with more perfect methods of sterilizing the vagina there was not the same danger of infecting the peritoneum as formerly. The next step was to secure the uterine arteries on each side either by ligatures or clamp forceps. If ligatures were used, a curved needle threaded with silk was passed over the uterine artery close to the cervix on either side and tied on the vaginal aspect; the ends of the ligature were left long. The ligatures were passed close to the cervix so as to be well within the half-inch of tissue between the ureter and the cervix, to avoid wounding the former structure. If clamps were used they were left on. Then the cervix was separated by cutting on the uterine side of the ligatures and the growth and cervix were cut off with scissors as high up as the bladder had been separated, the section often being cone-shaped with point upwards so as to remove as much uterine tissue as possible. Bleeding points, if any, were picked up and tied, and finally the cavity above and the vagina were packed, in the earlier cases with lint soaked in carbolic oil, but in the later cases with iodoform gauze. As a rule the packing was removed in one or two days and replaced once, to be followed by antiseptic douching of the vagina until the wound had granulated up. Clamps, if used, were removed in thirty or forty-eight hours, the shorter time seemed to be just as useful as the longer for securing hæmostasis. The ligatures usually came away in

three or four weeks by themselves, but sometimes had to be removed by traction.

*Vaginal Hysterectomy* was performed sixty-two times. The usual method of operating was as follows:—The vulva was shaved and the vagina and buttocks rendered as aseptic as possible, and the patient placed in the lithotomy position. With a speculum holding down the posterior vaginal wall the cervix and growth were seized with tenacula. After ascertaining the position of the bladder on the cervix by means of a sound an incision was made in front as wide of the growth as possible, exactly as in the supra-vaginal amputation. Next the bladder was separated from the anterior surface of the uterus as far as the utero-vesical pouch of peritoneum. The peritoneum was opened here, either by pushing the finger through or snipping with scissors. This opening was torn widely from side to side with the fingers. Next an incision was made behind the cervix, connecting the ends of the anterior incision. This cut opened the pouch of Douglas as a rule, and when completed from side to side left the uterus only attached by the broad and round ligaments. The edges of the cut peritoneum should here be seized with forceps, so as to prevent any unnecessary stripping up of the pelvic peritoneum from the cellular tissue. The next step was to secure the vessels in the broad ligament. Clamps or ligatures were used for this in the same way as in supra-vaginal amputation. Then the lower part of the uterus was freed from the broad ligaments by cutting with scissors on the uterine side of the clamps or ligatures. These cuts also separated the attachments of the utero-sacral ligaments to the uterus. Next the upper parts of the broad ligaments were dealt with. If they were ligatured, the fundus uteri was first retroverted and brought into the vagina through the posterior incision, so that the broad ligaments could be seen. These were then transfixed with a needle threaded with silk and tied in two or more places, as was necessary. Finally the uterus was removed by cutting on the uterine side of the ligatures, which were left long. If clamps were used, either the large Doyen broad ligament forceps or many pairs of Spencer Wells' forceps were made use of. In

fixing the clamps great care was taken that no bowel or other structure than the broad ligament was included. As a rule one half of the uterus was cut free first, leaving the other broad ligament much easier to deal with. Occasionally ovaries and Fallopian tubes had to be removed at this stage if any obvious disease in them was found. This was easily done by including them in the clamps or ligatures. The operation was completed either by simply plugging the vagina and lower part of the pelvic cavity with gauze, or by first bringing the pelvic peritoneum together with a few sutures and then plugging the vagina. Either method seemed to answer equally well. The clamps were removed in thirty to forty-eight hours, and ligatures, always left long, were removed or came away as a rule within four weeks. The gauze plugs were usually renewed once or twice, and then gentle irrigation of the vagina was practiced, from about the fourth day onwards. Vaginal hysterectomy completed by opening the abdomen from above was performed twice and abdominal pan-hysterectomy once. None of these operations require a separate description. The first two operations were completed by the abdominal route because of unusual difficulties encountered in finishing by the vagina. (Cases 81 and 91). Abdominal pan-hysterectomy was performed because of the large size of the uterus in Case 102.

The difficulties encountered in performing these operations were :—

1. The difficulty of drawing down the uterus on account of infiltration of the tissues around by growth, or by old cicatrices.
2. Mechanical difficulties owing to the small size of the vagina.
3. Difficulty of getting a firm hold with the tenacula on account of soft friable growth.
4. Difficulty of separating the bladder when the growth was spreading anteriorly.
5. Difficulty of retroverting the uterus owing to the large size of the fundus. Sometimes this was due to pyometra, and once at least to the presence of old fibroids.
6. Difficulty of delivering the fundus owing to its large size and great length.

7. Difficulty of applying clamps in cases where the uterus was large or very elongated.

8. Difficulties with old adhesions between the fundus uteri and neighbouring organs.

These difficulties need no special description, and the means of overcoming them depends largely on the skill of the individual operator.

The accidents attending this series of operations were very few indeed. There were no cases of primary hæmorrhage of any importance, and only two of secondary hæmorrhage, in Case 27 on the first day after operation, and Case 71 on the eighth day. Both were arrested by plugging, but Case 27 died of peritonitis. There were two cases of vesico-vaginal fistula, Cases 77 and 83. Both fistulæ were discovered some days after the operation, and in both clamps were used for the lower parts of the broad ligaments. It was never discovered whether the fistulæ were caused by damage during the operation, or by the clamps. Both cases recovered from the immediate effects of the operation but the fistulæ remained open. In no case was there any injury to the ureter, or to intestine or other organ by the clamps.

The progress of the cases after operation in most instances was uneventful. Apart from those who died, very few cases had pyrexia, although a good many had some suppuration of the vaginal wound. This it is almost impossible to prevent, however carefully the cases are douched. One case, number 54, had an abscess in the cellular tissue above the vagina, which eventually opened into the vagina and did well. In one case, number 91, there was very severe septic infection, accompanied by peritonitis, bronchitis, diarrhoea and sloughing of the abdominal wound; this was one of the cases in which the abdomen was opened above to complete the operation. Ultimately she did well, and is alive now, three years after the operation.

*Immediate results of the operations.* — Altogether there were thirteen deaths in the 102 cases, nearly 18 per cent. If the cases are divided into two halves, the mortality of the first fifty cases was eight, or 16 per cent., while that of the next fifty-two cases was five, or nearly 10 per cent. This no doubt

is to be accounted for by the fact that in later years the methods of preparing the patient and rendering the genitals aseptic were much more perfect, and so the risk of infection was nothing like so great. Also no doubt the improved means for sterilizing instruments and the operator's hands contributed something to this difference of mortality. Of the thirteen deaths, ten were from sepsis certainly, they were Cases 1, 28, 31, 39, 40, 42, 56, 81, 84, 87, and all are stated in the post-mortem records to have had peritonitis. One at least, number 56, could scarcely have been prevented, for there were pus cavities in the uterine wall, which gave way during the operation and no doubt infected the peritoneum. Cases 81 and 87 were exceptionally bad ones for operation and must have been well nigh impossible to disinfect. One case, number 2, died apparently from collapse after one of her numerous operations. Number 26 died from emphysema of the lungs, no other lesion being found. Number 66 rendered very anæmic by the growth and operation died of gangrene of the lung, no other lesion being discovered.

In the later years hysterectomy by the vaginal method was performed more extensively than supra-vaginal amputation, and the death-rate was distributed thus: In the first fifty-five cases supra-vaginal amputation thirty-two times with three deaths, vaginal hysterectomy twenty-three times with five deaths. In the last forty-seven cases supra-vaginal amputation four times with one death, vaginal hysterectomy thirty-nine times with three deaths; the percentage being, vaginal hysterectomy sixty-two cases with eight deaths, 13 per cent. nearly; supra-vaginal amputation thirty-six cases, with four deaths, or 11 per cent. about. So that apparently the mortality of the lesser operation may be expected to be very little less than that of the greater.

The reasons for the greater preponderance of hysterectomies in the later cases are not far to seek. It was found that the operator could not always be sure how far up the uterus a growth extended, and it was often surprising to see how a comparatively small vaginal growth had in some instances reached the internal os. Such cases would inevitably only be half treated

if amputation alone were performed. Nowadays, too, it is regarded universally elsewhere as a surgical principle, that organs with malignant growths should be widely removed, and it seems bad surgery to amputate a cervix and leave some possible growth in the body of a uterus. Vaginal hysterectomy is a more difficult operation to perform than supra-vaginal amputation, owing to the difficulties encountered in dealing with the upper parts of the broad ligaments. The opening of the utero-vesical pouch of peritoneum and ligaturing or clamping the upper parts of the broad ligaments, is always the most difficult part of the operation. Various modifications of the operation have been suggested of late years, which either attempt to make the operation easier or deal with the question of hæmostasis. The most important modification in the former category is the method of Howard Kelly, and consists in the bisection of the uterus in the mid-vertical line with scissors, before dealing with the broad ligaments. This procedure the author claims to cause but little hæmorrhage, because it is well known that the vessels in the mid line of the uterus have but small anastomoses. When the uterus is completely bisected, it is obvious that it is not only easier to deal with the broad ligaments by clamp or ligature, but it is possible to remove the growth more widely without damage to the ureters or surrounding structures. Especially is this latter possibility the case, if Kelly's method of cathetrisation of the ureters is first performed. Then the ureter, stretched on a catheter, can be readily felt and seen, and as readily dissected free from growth, or even a piece of it deliberately excised with the growth, the ends being brought together afterwards, or the proximal end turned into the bladder.

As far as hæmostasis is concerned, the clamp or the ligature are still most commonly used in this country.; but on the continent methods aiming at crushing the tissues of the broad ligament, by specially constructed very powerful forceps, are now in vogue. These forceps require to be only left on the tissues a few minutes and do undoubtedly secure perfect hæmostasis. Cases have, however, occurred where hæmorrhage has occurred immediately the clamp or *pincers à pression* were removed. At



present this method has found but little support in Great Britain. Some operators abroad are also aiming at the abolition of vaginal hysterectomy in favour of abdominal, on the grounds that much more "sweeping" operations can be performed by the latter route. For the reasons given in the earlier paragraphs, and also for the fact that these operations must be attended by a greater immediate mortality, they have not yet been widely performed in this country.

*The after-history of the Cases.*—This, always difficult to get at satisfactorily, has been no exception to the general rule, because 29 of the 102 cases cannot be traced at all by ordinary methods. The method used in this instance was to send reply-paid post-cards to all the patients at their original addresses. Of these, definite answers were obtained in seventy-three instances, the rest—twenty-nine—could not be traced. Of these cases, seventeen had no recurrence for periods varying from two to eleven years, and sixteen are now alive in May, 1901. One lived ten years and eventually died of cerebral disease, but had no recurrence of growth.

Of these sixteen cases :—

1 has lived 11 years since the operation.

1	"	10	"	"
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1	"	6	"	"
---	---	---	---	---

3	"	5	"	"
---	---	---	---	---

6	"	3	"	"
---	---	---	---	---

4	"	2	"	"
---	---	---	---	---

Thirty-one cases died of recurrence of growth at variable times after the operations :—

1 died of recurrence 5 years after operation.

1	"	4	"
---	---	---	---

2	"	3	"
---	---	---	---

3	"	2	"
---	---	---	---

12	"	between 1 & 2	"
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2	"	11 months after operation.
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4	"	10	"
---	---	----	---

1	"	9	"
---	---	---	---

2	"	8	"
---	---	---	---

1	"	7	"
---	---	---	---

2	"	5	"
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Twelve cases are known to have had recurrence of growth soon after the operation, one is alive still, four years since the operation; one is alive after two years and has a recurrence. The other ten cases are probably all dead, but the dates of their deaths cannot be ascertained.

Twenty-nine cases cannot be traced, and thirteen died as an immediate result of the operation, and thus the 102 cases are accounted for.

Very few conclusions can be drawn from these results, because there is not the slightest doubt that a very large number of the cases were very bad ones for operation, and it was only with a very laudable desire to try to do something for these patients, that the operations were undertaken. Even in these bad cases the operation is often justifiable, because the patients do not seem to suffer so much pain with recurrent growths as with the original, and because many have periods of comparative comfort after the operation who must have suffered enormously if nothing had been done for them. In answering the post-cards several stated that the operation was of great benefit to the patient although recurrence ultimately took place. Also the twenty-nine cases which could not be traced spoil the value of the series from a statistical point of view. The great fact that these results point to is this, that no operator can hope to get good results in cases of uterine cancer unless he gets his cases early.

The only way to get cases of cancer early is for all to realise the importance of immediate investigation of all cases of irregular bleeding in women; and particularly in women near the menopause, where irregular hæmorrhages are only too likely to be put down to "change of life" without proper investigation. The only way to be sure is to examine every patient at once who complains of bleeding, and if the slightest doubt exists as to the condition of a cervix or endometrium, to remove pieces with a curette and have them examined microscopically. In conclusion, I have to thank Dr. Galabin and Dr. Horrocks for their permission to publish and make use of their cases.

LIST OF CASES.

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CASE 1.—C. B., æt. 35. Dr. Galabin, Dorcas 8, July 13th, 1886. Married; one child, eleven years previously. Admitted for vaginal discharge. Had had yellowish discharge for two years, getting worse for nine months previous to admission. During this time discharge tinged with blood. Had lost flesh. Micturition sometimes involuntary.

*On examination.*—A mushroom-shaped growth protruding into the vagina. No cervical canal found; uterus somewhat fixed.

*Operation.*—Supra-vaginal amputation of cervix. The lower parts of the broad ligaments were secured with ligatures before cutting away the cervix. Cauterization afterwards with perchloride of iron. Vagina plugged with lint and carbolic oil.

*Parts removed* showed that the incision was above the growth, but not quite clear on one side.

*Progress of case* was not satisfactory. First there was some secondary hæmorrhage, then the discharge became very offensive and is noted as "feculent." Finally the patient died with signs of peritonitis.

*Autopsy* shewed that the rectum and Douglas' pouch were opened, and that both iliac veins were thrombosed. Some peritonitis was found. The cause of death was no doubt septicæmia.

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CASE 2.—L. S., æt. 18. Dr. Galabin, Dorcas 15, October 5th, 1886. Single. Menstruation regular, five to six days duration, four weeks interval. Admitted for a blood-stained vaginal discharge. Had previously been under Dr. Galabin in 1884 with a pendulous growth of the cervix which was removed and pronounced to be a "large celled mixed sarcoma." After the first operation she remained well until two months before this admission.

*On examination*, a large soft gelatinous mass filling up the vagina was found. It sprang from the anterior part of the cervix, and bled freely on touching.

*Operation.*—The growth first removed with a snare, and then at a second operation the cervix was amputated. The hæmorrhage was controlled by plugging with perchloride of iron. Later the mass was found to have grown again, and was again removed with the écraseur and chloride of zinc applied on tampons. No description of parts removed except that the growth was a mixed celled sarcoma. Recovery.

*After-history*—Admitted August, 1887. More partial removals were performed; after one of which operations the patient died rather suddenly, apparently from collapse.

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CASE 3.—L. C., æt. 49. Dr. Horrocks, Dorcas 23, April 27th, 1886. Married, five children, the last pregnancy fourteen years previously. Two months before admission a red discharge commenced, later this became continuous and more hæmorrhagic.

*On examination*, there was a growth found, which was most prominent on the posterior lip of the os uteri. It was ulcerated and bled very easily. Some impairment of mobility of the uterus. No pain nor wasting. No micturition troubles.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Supra-vaginal amputation. Ligatures used to arrest hæmorrhage. Peritoneum not opened.

*Parts removed* showed that the incisions had gone through growth at the sides.

*Progress*.—Uneventful. Recovery.

*After-history*.—Patient died soon after the operation, date not known, apparently the growth continued uninterruptedly.

CASE 4.—E. S., æt. 43. Dr. Galabin, Dorcas 14, April 12th, 1886. Married, four children, two miscarriages; the last pregnancy seven years before admission. Admitted for blood-stained discharge and growth of cervix. Seven months before admission the menstrual periods became prolonged and the loss greater than usual. Four weeks before admission a discharge, which was yellowish or blood-stained, continued after her monthly period. Had lost some flesh, and had a pain in the left side. No micturition troubles.

*On examination*.—Cervix found to be involved throughout in growth. Ulcerated on anterior portion, bathed in very foul discharge.

*Diagnosis*.—Carcinoma of uterus.

*Operation*.—Supra-vaginal amputation, apparently without first ligaturing the uterine arteries.

*Parts removed* showed that the growth did not extend deeply into the cervix, and was entirely removed.

*Progress of the case* was satisfactory. Recovery.

*After-history* not to be traced.

CASE 5.—M. S., æt. 32. Dr. Horrocks, Dorcas 22, September 21st, 1886. Married, four children, one miscarriage; last pregnancy eleven months previously. Admitted for carcinoma of cervix, with pregnancy at the sixth month. Fourteen days before admission hæmorrhage came on, and continued. Induction of labour was performed with a tedious delivery by craniotomy, owing to non-dilatation of the cervix.

*On examination*.—The cervix was split, and some hard distinct nodules of growth were felt in it.

*Operation*.—Supra-vaginal amputation.

*Progress* uneventful. Recovery.

*After-history* cannot be traced.

CASE 6.—J. M., æt. 34. Dr. Galabin, Dorcas 7, January 14th, 1887. Married, four children, two miscarriages; the last pregnancy ten months previously (a four months' miscarriage). Admitted for flooding. Had been regular until twenty-two months before admission, when she began to have frequent hæmorrhages. Fourteen months before admission she became pregnant, but the hæmorrhages did not cease. She miscarried at the fourth month. Since then the hæmorrhages had been of weekly occurrence.

*On examination.*—The margins of the os uteri were found to be rugged, ulcerated and everted, bleeding readily on touching. The cervix seemed enlarged as a whole, but was mobile. Broad ligaments were free.

*Operation.*—Supra-vaginal amputation. The lower parts of the broad ligaments were ligatured with silk.

*Parts removed* showed that the incision had gone through healthy tissues.

*Progress of the case* was rather slow, as the wound did not granulate well. Zinc chloride was applied, and eventually the patient went out. Recovery.

*After-history.*—Was re-admitted on April 17th with a recurrent growth. This was treated with chloride of zinc, and the patient went out *in statu quo*.

CASE 7.—I. M., æt. 39. Dr. Galabin, Dorcas 14, November 25th, 1887. Married, eleven children, the last eleven months previously. Admitted for bleeding from uterus. Had had no menses for four years, presumably on account of pregnancies. Three months before admission began to have hæmorrhages, which later became profuse. No pain, no micturition troubles, no wasting.

*On examination*, a mass of growth as large as a walnut on posterior lip of cervix was found, coarsely granular in appearance, and bled readily on touching.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation, ligatures used for the uterine arteries.

*Parts removed* showed that the incisions had gone a quarter of an inch beyond the limits of all apparent growth.

*Progress* was marred by some hæmorrhage directly after the operation, which was arrested by plugging with wool and crystals of perchloride of iron. Later there was an attack of cystitis. Recovery.

*After-history.*—Patient re-admitted five weeks later with a recurrence. This was burnt out with the actual cautery and pledgets of wool soaked in chloride of zinc (300 gr. ad ʒi.) were applied. Eventually patient went out with a roughly granular cavity, which did not bleed on examination, in the site of the cervix. After this patient could not be traced.

CASE 8.—M. D., æt. 53. Dr. Horrocks, Dorcas 20, January 28th, 1887. Married, one child, one miscarriage, the last pregnancy twenty-seven years previously. Menopause at 38 years of age. Had suffered from prolapse of the uterus for many years, which had been treated with a ring pessary. Twelve months before admission she began to have an offensive vaginal discharge. Later hæmorrhage came on at intervals.

*On examination*, a growth was found springing from posterior lip of the cervix.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation.

*Parts removed* showed malignant growth, doubtful if it was entirely removed.

*Progress* uneventful. Recovery.

*After-history.*—Not to be traced.

CASE 9.—M. H., æt. 50. Dr. Galabin, Dorcas 15, January 3rd, 1888. Married, no children. Menopause five and a-half years before. Had had a

white discharge for five years, and two and a-half months before admission, hæmorrhages came on which continued more or less. No pain. No micturition trouble. Some wasting, though patient was a very stout woman.

*On examination*, a growth was found involving chiefly the anterior lip of the cervix. There was some bleeding on examination.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Supra-vaginal amputation. Difficult owing to the nulliparous vagina. Hæmorrhage was arrested by strips of lint soaked in tincture of perchloride of iron.

*Parts removed* shewed that the incisions had gone beyond the limits of the growth.

*Progress* uneventful. Recovery.

*After-history*.—Died in May, 1898, of "Softening of the Brain," Dr. Kelsey, of Red Hill, kindly supplied the information that she had no return whatever of the growth.

CASE 10.—H. V., æt. 45. Dr. Galabin, Dorcas 11, April 14th, 1888. Married, six children, the last twenty-two years previously. Admitted for bleeding from the vagina. Menses were regular, four-weekly, lasting four or five days, with considerable loss. For twelve months had been losing almost continuously, except for one period of three weeks. There was no pain and no loss of flesh.

*On examination*, a growth was found extending up the cervix, more particularly to the left. It was found that the base of the bladder was infiltrated, and also the peritoneum in Douglas' pouch.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Amputation of cervix.

*Parts removed* showed that the whole of the growth had not been removed.

*Progress* uneventful. Recovery.

*After-history* cannot be traced.

CASE 11.—E. C., æt. 27. Dr. Galabin, Dorcas 13, June 17th, 1889. Married, seven children, two miscarriages, the last pregnancy three years previously. Admitted for pain and offensive discharge. Menses regular until three months before admission. History seemed to date back twenty months before admission, from a flooding which occurred at that time. Discharge offensive for three months.

*On examination*, a mushroom-shaped growth, with an everted edge, bleeding readily, was found. It appeared to reach the vaginal mucous membrane on the right side. No growth felt in cellular tissue outside cervix.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Supra-vaginal amputation. Ligatures for broad ligaments.

*Parts removed* showed that there was some doubt as to whether all the growth had been removed posteriorly.

*Progress* was uneventful. Recovery.

*After-history*.—The growth recurred, and the patient died in February, 1891.

CASE 12.—S. F., æt. 38. Dr. Galabin, Dorcas 9, February 18th, 1889. Married, seven children, the last eight years before. Admitted for bleeding. One year before admission the monthly periods began to lengthen in duration,

and a slimy discharge appeared. Later, the periods lasted a fortnight, and in addition there was a very foul sanguineous discharge. No pain, some slight degree of wasting.

*On examination*, the cervix was found to be bilaterally split, and covered with irregular prominences, which bled on touching.

*Diagnosis*.—Carcinoma of the cervix.

*Operation*.—Supra-vaginal amputation.

*Parts removed* showed that the incisions were wide of the growth everywhere.

*Progress* uneventful. Recovery.

*After-history*.—Not to be traced.

CASE 13.—G. F., æt. 39. Dr. Galabin, Dorcas 12, February 4th, 1889. Married, eight children, three miscarriages, the last pregnancy two years previously. Menses regular, four-weekly, lasting three days; no great loss. Six months before admission had a yellowish discharge tinged with blood, which soon became offensive. Micturition painful. Lost flesh for three months.

*On examination*, a growth of a mushroom shape found on anterior lip of cervix. Some bleeding on examination. Uterus was free and movable.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Supra-vaginal amputation of cervix.

*Parts removed* showed that the growth was confined almost entirely to the anterior lip of the cervix.

*Progress* uneventful. Recovery.

*After-history*.—Not to be traced.

CASE 14.—A. M. M., aged 36. Dr. Galabin, Dorcas 18, June 26th, 1889. Married, six children, the last seven years previously. Menses regular until ten months previously, when they varied as to interval. Sometimes the interval would be three weeks, at other times four or five weeks. Also, during this time the amount lost had been greatly increased, and on two or three occasions, just before admission, was profuse. Had had pain referred to the uterus, of a pricking nature. No wasting or micturition troubles.

*On examination*, a papillary looking growth was found projecting from the thickened anterior lip of the cervix. This bled on examination. Uterus was movable.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Supra-vaginal amputation. Ligatures used for the broad ligaments.

*Parts removed* (?).

*Progress* was uneventful, but when discharged there seemed to be a nodule in the stump of the cervix. Recovery.

*After-history*.—Was re-admitted in June, 1890, with a recurrent growth spreading into base of right broad ligament. Inoperable. Not to be traced after this.

CASE 15.—A. O., æt. (?). Dr. Galabin, Dorcas 16, May 30th, 1889. Married, seven children. Admitted for hæmorrhage and loss of flesh. Menses had been regular until seven months before admission, and then she noticed

that the loss continued between the periods, with a day or two interval occasionally. Never any severe flooding. No pain and no other discharge.

*On examination* the cervix was found expanded by growth and ulcerated on the surface. On the left side the growth nearly touched the vaginal wall. Bled readily on touching.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation. Ligatures used for the uterine arteries.

*Parts removed* showed that the growth extended one and a-half inches up the cervical canal, and that the incisions apparently had gone well beyond everywhere.

*Progress*, except for some hæmorrhage on the first two days, was uneventful. Recovery.

*After-history.*—Not to be traced.

CASE 16.—H. V., æt. 38. Dr. Galabin, Dorcas, 10, March 22nd, 1889. Married, ten children, one miscarriage, the last pregnancy seven months previously. After this confinement the patient lost more or less continuously until three weeks before admission, when she had two severe hæmorrhages, with a week's interval.

*On examination*, the cervix admitted the finger, which entered a rugged cavity, bleeding readily on touching. Cervix somewhat expanded.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy, ligatures applied to broad ligaments. No difficulty.

*Parts removed.*

*Progress* uneventful. Recovery.

*After-history.*—Recurrence took place and death occurred in December, 1890.

CASE 17.—F. W., æt. 45. Dr. Galabin, Dorcas 13, May 20th, 1889. Married, four children, two miscarriages, the last pregnancy eight years previously. Menses natural until eight months before admission, when she had a severe hæmorrhage at the monthly time lasting three weeks. After this the discharge was continuous, watery, and brownish. Pain five weeks before admission. No micturition troubles.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation.

*Progress* uneventful. Recovery.

*After-history.*—Recurrence of growth occurred, and the patient died in May, 1893.

CASE 18.—A. G., æt. 66. Dr. Galabin, Dorcas 6, June 10th, 1890. Married, 16 children, the last twenty-two years previously. Admitted for pain and blood-stained discharge. Menopause occurred at 51. No discharge complained of until two years before admission, when it was slightly blood-stained, and small in amount. A few months before admission noticed a hard lump in the vagina when using a syringe. Pain in the loins complained of.

*On examination.*—Cervix enlarged, hard and irregular. Growth encroaching on vaginal wall somewhat on right side. Fundus not enlarged. Uterus movable as a whole. No bleeding on examination.



*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Ligatures used for broad ligaments. Bladder separated with much difficulty.

*Parts removed* (?).

*Progress* uneventful. Recovery.

*After-history.*—Growth recurred and death took place twelve months after the operation.

CASE 19.—L. W., æt. 27. Dr. Galabin, Dorcas, 16, May 27th, 1890. Married, three children, the last, five months previously. Three months before admission a white discharge commenced, which soon became bright red, and continued so. Had some pain on walking. Lost no flesh.

*On examination* the uterus was somewhat fixed, the anterior lip of the cervix was hard and rolled upwards. Did not bleed readily.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Broad ligaments ligatured.

*Parts removed* (?).

*Progress* uneventful. Recovery.

*After-history.*—Not to be traced.

CASE 20.—J. A., æt. 48. Dr. Galabin, Dorcas 14, September 21st, 1891. Married, three children, the last seventeen years previously. Menopause occurred at 45. Seven years previous to her admission had been operated on for a profuse red discharge, which then ceased until three months before admission. Then it was accompanied by a pain in the left side. Pain before micturition, and loss of flesh.

*Condition on admission* (?).

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation, no difficulty. Ligatures used for the lower ends of the broad ligaments.

*Parts removed* (?).

*Progress* was satisfactory, but patient seemed to have become "light-headed" during her convalescence. Recovery.

*After-history.*—Not to be traced.

CASE 21.—A. C., æt. 48. Dr. Horrocks, Dorcas 21, June 16th, 1890. Admitted for flooding and constant loss. Married, five children, the last, fourteen years previously. Two years before admission the monthly periods, which had been regular and normal, stopped for three months. Then they came on again, and since that time she continued to lose blood, and discharge tinged with blood (twenty-one months). She had had pain for three months before admission.

*On examination*, the cervix was found small, irregular and fungating. It bled readily on touching.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Ligatures used to control the broad ligament vessels.

*The parts removed* showed that the growth extended up to the internal os uteri.

*Progress* was uneventful. Recovery.

*After-history.*—Died of recurrent growth in October, 1891.

**CASE 22.**—M. L., æt. 45. Dr. Horrocks, Dorcas, 6, July 17th, 1890. Married. Complained of pain; bleeding from vagina and foul discharge. How long the symptoms lasted was not stated in the report.

*On examination*, a mass of growth starting from the cervix was found.

*Operation.*—Supra-vaginal amputation.

*Parts removed* (?).

*Recovery.*

*After-history.*—Growth recurred and the patient died in May, 1891.

**CASE 23.**—M. P., æt. 43. Dr. Horrocks, Dorcas 9, August 5th, 1890. Married, four children, the last four years previously. Menses regular until seven months before admission. During this time there had been a foul discharge from the vagina. Had been losing flesh. Micturition very frequent.

*On examination.*—Large mass of growth starting from cervix. Vaginal walls not infiltrated. Bleeding on examination. Had been judged too advanced for operation in another hospital.

*Operation.*—Supra-vaginal amputation of the cervix.

*Parts removed* seemed to have been cut well beyond the limits of the growth.

*Progress* uneventful. *Recovery.*

*After-history.*—Seen at out-patients' in January, 1899, there was no recurrence of the growth. Reports herself as alive but not in very good health in May, 1901.

**CASE 24.**—M. E., æt. 61. Dr. Galabin, Dorcas 15, April 20th, 1891. Married, eight children, the last eighteen years previously. Menses appear to have been regular in spite of her age until one year before admission. Then the intervals became shorter and the amount of blood lost greater. The discharge was often offensive.

*On examination*, a ragged cavity was felt inside the anterior lip of the cervix. The uterus was not very freely movable. Pieces of growth came away on examination, with free bleeding.

*Diagnosis.*—Carcinoma of cervix (confirmed by microscope).

*Operation.*—Vaginal hysterectomy, clamps and ligatures being used.

*Parts removed* shewed the ragged cavity and growth of cervix above mentioned. Also a sub-peritoneal fibroid and one sub-mucous fibroid. The uterus measured five inches. These fibroids, no doubt, accounted for the delayed menopause.

*Progress* was interrupted by an attack of cystitis and a bed-sore, otherwise good. The ligatures came away on the ninth day. *Recovery.*

*After-history.*—May, 1901, is reported to be quite well and has no recurrence of growth.

**CASE 25.**—M. F., æt. 52. Dr. Galabin, Dorcas 9, October 8th, 1891. Married, two children, one miscarriage. The last pregnancy eighteen years previously. Admitted for pain and hæmorrhage. Menopause seven years before admission. No discharge until twelve months before admission, then discharge came on, hæmorrhagic and mucous in character. This continued with more free hæmorrhage occasionally. Pain complained of twelve months, smarting on micturition and some loss of flesh.

*On examination*, the cervix was found to be eroded, the mobility impaired, and bleeding occurred on examination.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation. Ligatures used for the broad ligaments.

*Parts removed* shewed that the uterine cavity was dilated and contained pus. No account of the growth.

*Progress* uneventful. Recovery.

*After-history.*—The growth recurred and the patient died in March, 1893.

CASE 26.—S. H., æt. 65. Dr. Galabin, Dorcas 8, July 17th, 1891. Married, two children, the last thirty-three years before. Admitted for pain and hæmorrhage. Menopause occurred at 52, and from that time until about three months before admission there was no discharge. Then hæmorrhages began and continued until her admission. No condition on examination in the report.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Ligatures used for the broad ligaments.

*Parts removed* not described.

*Progress* unsatisfactory from first. Only slight rise of temperature, but pulse never came down below 100 after the operation.

*Died* on the fourth day.

*Autopsy.*—Beyond emphysema of both lungs, no lesion was found to account for death. There were no secondary growths.

CASE 27.—L. T., æt. 41. Dr. Horrocks, Dorcas 21, April 13th, 1891. Admitted for bleeding. Married, one child, one miscarriage; the last pregnancy twenty months previously. Had suffered from cervical erosion years before. Menses regular, always copious until three months before admission, when the bleeding became continuous. There had been wasting and pain, gradually getting worse. No micturition troubles.

*On examination.*—Both lips of cervix were rough and much thickened. A portion removed for microscopical examination showed epitheliomatous growth.

*Diagnosis.*—Squamous epithelioma of cervix.

*Operation.*—Vaginal hysterectomy. Lower parts of broad ligaments clamped, upper parts ligatured.

*Progress.*—Severe hæmorrhage occurred the same evening as the operation. This was controlled by firm plugging of Douglas' pouch. The temperature gradually rose to 102°, and patient died on the third day, with signs of peritonitis.

*Autopsy* revealed nothing to cause death.

CASE 28.—S. S., æt. 43. Dr. Horrocks, Dorcas, 21, December 28th, 1891. Admitted for hæmorrhage and pain. Married, no children. Had been a widow seven and a-half years. Always had been regular until three years before admission. Then the periods began to be prolonged, and losses occurred between the periods as well.

*Diagnosis.*—Carcinoma of the cervix.

*Operation.*—Supra-vaginal amputation. No difficulty.

*Progress.*—Five days after the operation the temperature went up to 104°, and the next day the patient had two rigors. Headache, abdominal distension

and diarrhoea occurred at the same time as the rigors. Then came cystitis and also a painful eye, which, however, was not caused by the general septicæmic process. The patient became worse, œdema of the legs being the last symptom noted, and death occurred thirteen days after the operation.

*Autopsy.*—Vegetations on the mitral valve, and infarcts in spleen and kidneys; not suppurating. Some suppuration at the site of the amputated cervix, and also a vein in the pelvis contained pus. This case no doubt ended as an acute septicæmia.

CASE 29.—M. B., æt. 41. Dr. Galabin, Dorcas 20, September 5th, 1892. Married, seven children, two miscarriages; the last pregnancy two years previously. Admitted for pain and intermenstrual discharge. Had noticed intermenstrual hæmorrhages for about twelve months, and during that time the menstruation had been profuse, lasting seven days. Pain had been present for two months. Micturition frequent; no wasting.

*On examination.*—Cervix irregular and eroded, bled on examination. Portions of the eroded cervix removed for microscopic examination showed squamous carcinoma.

*Operation.*—Vaginal hysterectomy. No great difficulty, although the uterus could not be very well drawn down. Ligatures used for the broad ligaments.

*Progress* was delayed on account of some considerable pyrexia caused by sepsis, and attended by alkaline urine, joint pains, and much flatulence. This, however, gradually cleared up. Recovery.

*After-history.*—Died in April, 1893, of recurrence of growth.

CASE 30.—E. G., æt. 39. Dr. Galabin, Dorcas 9, October 26th, 1892. Married, two children, three miscarriages; last pregnancy two years previously. Admitted for hæmorrhage. Began to have irregular bleeding and bleeding on coitus two months before admission. No other discharge noted.

*On examination.*—The whole circumference of the cervix was broadened and ulcerated, with everted edges.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation. Ligatures used for vessels.

*Parts removed* measured two and a-half inches below in diameter, and was two inches vertically. The cut surface was quite above the line of the growth.

*Progress* uneventful. Recovery.

*After-history.*—Not to be traced.

CASE 31.—D. H., æt. 42. Dr. Galabin, Dorcas 12, January 7th, 1892. Married, four children, the last ten years previously. Admitted for continuous hæmorrhage. For twelve months patient had been suffering from a brownish watery discharge with hæmorrhage constantly recurring. Was never more than two days free. Menstruation could not be distinguished as such during this time. Some occasional pain and progressive wasting. No micturition troubles.

*On examination*, a large mass of growth was found springing mostly from the posterior lip. Much bleeding on examination. No impairment of mobility of the uterus.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation after removal of the growth with the écraseur.

*Parts removed* showed that the incisions had apparently completely removed the growth.

*Progress* was unsatisfactory from the first.

*Death* occurred on the third day after operation, with signs of peritonitis.

*Autopsy* showed general peritonitis, with much lymph production and matting of intestines. The growth also had not quite been all removed by the operation.

CASE 32.—H. T., æt. 53. Dr. Galabin, Dorcas 18, February 15th, 1892. Married, two children, one miscarriage. The last pregnancy twenty-five years ago. Menopause five years before admission. Had had a white vaginal discharge for many years; four months before admission this changed to a dirty brown watery discharge, and continued. There was very little pain, some wasting, no bowel or micturition troubles.

*On examination*, a growth of the cervix was found which extended a little on to the anterior vaginal wall, and ran up the cervical canal as far as the finger could reach.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy, ligatures being used for the broad ligaments.

*Progress* uneventful. Ligatures came away at the end of four weeks. Recovery.

*After-history.*—Not to be traced.

CASE 33.—S. D., æt. 50. Dr. Horrocks, Dorcas 23, January 26th, 1892. Admitted for pain and discharge. Married, 9 children, two miscarriages, the last pregnancy six years previously. No regular monthly period for thirteen months before admission. During that time had a coffee coloured discharge, accompanied by pain in the back and across stomach. Micturition frequent, but no incontinence.

*On examination*, a growth of the cervix which implicated the anterior vaginal wall was found.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation. Spencer Wells' forceps used to control uterine arteries and left on.

*Parts removed* showed that the incisions had gone very close to the growth at one part.

*Progress* uneventful. Recovery.

*After-history.*—Not to be traced.

CASE 34.—A. G., æt. 44. Dr. Horrocks, Dorcas 23, December 17th, 1892. Admitted for growth of the uterus. Married, three children, the last pregnancy seventeen years previously. Discharge commenced a year before admission; yellow in character. No hæmorrhages. The menstruation had been regular and lasted only three days with a four weeks' interval. Some wasting. Discharge offensive for six months. Micturition frequent.

*On examination*, the cervix was found thickened by growth, which bled readily on touching. Vaginal walls not invaded.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation. Vessels ligatured where bleeding.

*Parts removed* were not described.

*Progress* uneventful. Recovery.

*After-history.*—The growth recurred and the patient died in July, 1894.

CASE 35.—M. H., æt. 35. Dr. Horrocks, Dorcas 19, February 29th, 1892. Admitted for a "watery discharge." Married, five children, one miscarriage; the last pregnancy five years previously. The watery discharge commenced two years before admission, and a year later hæmorrhage took place at coitus. No offensiveness of discharge. Micturition normal. Menstruation unaffected.

*On examination*, a very large cauliflower mass of growth could be felt springing from both lips of the cervix. No invasion of the vaginal walls.

*Diagnosis.*—Epithelioma of the cervix.

*Operation.*—Supra-vaginal amputation. There was an unusual amount of bleeding, which was controlled by ligatures and hot douching.

*The parts removed* showed that the incision had gone well beyond the growth at all points.

*Progress of the case.*—Not quite satisfactory, as the temperature remained above normal for seven days. Recovery.

*After-history.*—Not to be traced.

CASE 36.—A. S., æt. 37. Dr. Horrocks, Dorcas 23, February 15th, 1892. Admitted for constant hæmorrhage. Married, two children; the last ten months previously. When pregnant with the last child patient had a fall, twelve months before admission. From that time she began to lose, and had continued more or less ever since. Sometimes offensive. No pain. Some wasting. Micturition not affected.

*On examination*, the cervix was felt to be hard and ulcerated, with a deep transverse fissure. This led into an excavation which bled freely on touching. Uterus movable. Several glands felt enlarged per rectum.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Silk ligatures used for the principal vessels, and five pairs of Spencer Wells' forceps also left on bleeding points. No difficulty.

*The parts removed* showed that the growth chiefly affected the anterior lip of the cervix, and had reached about to the level of the internal os.

*Progress* was uneventful. The ligatures came away on the eighteenth day. Recovery.

*After-history.*—Not to be traced.

CASE 37.—M. W., æt. 55. Dr. Horrocks, Dorcas 23, October 21st, 1892. Admitted for foul-smelling discharge. Married, two children; the last twenty-nine years previously. Menopause at 50. Had always had a discharge since the menopause (five years), but lost no blood until four months before admission. Since then it had been always blood-stained. Had lost flesh and suffered much pain. Micturition normal.

*On examination*, the cervix had a rugged cavity in it, with hard everted edges, bleeding readily on touching.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Ligatures, three on each side, being used to control the vessels in the broad ligaments. No difficulty.

*Progress* was uneventful. Ligatures had come away by the thirteenth day. *Recovery.*

*After-history.*—Growth recurred, and death took place in November, 1893.

CASE 38.—D. N., æt. 31. Dr. Galabin, Dorcas 13, November 4th, 1893. Married, three children, one miscarriage. The last pregnancy six years previously. Admitted for constant loss and pain. For three months before admission there had been constant hæmorrhage, so that the menstrual periods could not be distinguished. No pain, some wasting, no micturition troubles.

*On examination*, a mushroom-shaped growth was found, springing mostly from the posterior lip of the cervix. There was some induration in the left broad ligament, apparently due to an old laceration. Some induration on the upper third of the vagina on the left side.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Ligatures and clamps used for the broad ligaments.

*Parts removed* showed that the disease extended as high as the internal os uteri.

*Progress* uneventful; all the ligatures but one came away in three weeks, the last one in four weeks. *Recovery.*

*After-history.*—Not to be traced.

CASE 39.—S. C., æt. 57. Dr. Horrocks, Dorcas, 12, July 12th, 1893. Married, eight children, one miscarriage the last pregnancy, a miscarriage seven years previously. Admitted for constant discharge. Menopause ten months before admission. Discharge commenced nine months before admission, was at first yellow, afterwards streaked with blood. There had been hæmorrhage on coitus occasionally. Some pain was complained of, but no wasting. No micturition troubles.

*On examination*, a soft ulcerated growth was found at the site of the external os uteri, affecting mostly the posterior lip.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Ligatures used for the broad ligaments.

*Parts removed* showed that the incisions were well beyond the margins of the growth.

*Progress* was unsatisfactory from the first. Signs of peritonitis were present, the bowels did not act until the fourth day and there was much vomiting.

*Died* on 5th day.

*Autopsy* showed local pelvic peritonitis. No growth left behind.

CASE 40.—C. P., æt. 59. Dr. Horrocks, Dorcas 21, September 7th, 1893. Admitted for bleeding after coitus. Married, nine children, one miscarriage, The last pregnancy twenty-two years previously. Menopause at 52. Had been loosing flesh for four months. Nine weeks before admission had some hæmorrhage after coitus, which recurred at intervals, and also had a yellowish offensive discharge.

*On examination*, a growth was found within the cervical canal. Bled readily on examination.

*Diagnosis.*—Carcinoma of cervical canal. (Proved afterwards microscopically).

*Operation.*—Vaginal hysterectomy. Ligatures used for the broad ligaments and three pairs of Spencer Wells' forceps were also left on some bleeding points.

*Parts removed* showed that the growth was in the cervical canal, and had invaded the lower segment of the uterus. The cavity of the uterus was dilated and contained about an ounce of pus.

*Progress.*—The patient died on the fourth day, of peritonitis.

*Autopsy.*—Beyond commencing peritonitis there was nothing except some hæmorrhage into the posterior wall of the bladder found.

CASE 41.—E. V., æt. 47. Dr. Horrocks, Dorcas, 21, June 20th, 1893. Married, four children, one miscarriage, the last pregnancy seven years previously. Menstruation very scanty and irregular for three or four years. Twelve months before admission had bleeding after coitus, which rapidly increased in amount and was followed by more or less constant discharge. No pain or micturition troubles.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation.

*Progress* uneventful. The last ligatures were removed on the twenty-third day after operation. Recovery.

*After-history.*—Not to be traced.

CASE 42.—P. W., æt. 26. Dr. Horrocks, Dorcas, 21, December 11th, 1893. Admitted for profuse hæmorrhage from vagina. Single. Menstruation always regular until three months before admission when she had a profuse hæmorrhage. This continued until her admission more or less, with a watery discharge in the intervals between the hæmorrhages.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Ligatures were used to control the vessels in the broad ligaments. Operation difficult owing to the small size of the vagina.

*Parts removed* showed that the growth had extended up to the internal os, and had begun to infiltrate the posterior fornix.

*Progress.*—The temperature rose soon after the operation. Vomiting and abdominal distension occurred, and the patient died on the third day, with signs of peritonitis.

*Autopsy.*—Pelvic peritonitis was found with suppuration about the ligatures.

CASE 43.—S. B., æt. 56. Dr. Galabin, Dorcas, 6, March 5th, 1894. Single. Admitted for foul discharge from vagina. Menopause occurred at 50. One year before admission the discharge commenced, it was thick and often streaked with blood. Later it became foul smelling. Some backache the only pain mentioned. Lost flesh for six months before admission.

*On examination*, the cervix was found to be atrophied and the uterine cavity enlarged, the sound passed three and a-half inches. Epithelioma was diagnosed from curettings.

*Operation.*—Vaginal hysterectomy, difficult owing to the narrow vagina. Clamps and ligatures used.



*Parts removed* shewed that the uterus contained fibromyomata, which were becoming invaded with malignant growth from the endometrium.

*Progress* uneventful except for some delusions and delirium. Ligatures came away in three weeks. Recovery.

*After-history.*—In May, 1901, reports herself in extremely good health.

CASE 44.—M. C., æt. 44. Dr. Galabin, Dorcas 18, April 16th, 1894. Married, five children; the last pregnancy four years previously. Admitted for severe hæmorrhage. Twelve months before admission began to have irregular hæmorrhages, which continued up to the time of admission. There had been no pain, some loss of flesh for five months, and a continuous vaginal discharge.

*On examination*, the cervix was found to be infiltrated by growth, which bled readily on examination, and was soft and friable.

*Operation.*—Vaginal hysterectomy. Ligatures used for the broad ligaments.

*Parts removed.*—Not described.

*Progress* uneventful. Recovery.

*After-history.*—The growth recurred, and death took place in December, 1894.

CASE 45.—J. F., æt. 58. Dr. Galabin, Dorcas 18, November 7th, 1894. Married, nine children; the last, fourteen years previously. Twelve months before admission discharge of "thick matter," streaked with blood commenced. Later the discharge became almost entirely blood. No pain or wasting. Micturition frequent.

*On examination*, a papillary growth, which bled freely, found on the posterior lip of the cervix. Some impairment of mobility.

*Diagnosis.*—Carcinoma.

*Operation.*—Vaginal hysterectomy. Some difficulty in separating bladder, which was possibly implicated by the growth. Single clamps used for the broad ligaments.

*Parts removed.*—No description.

*Progress* uneventful. Recovery.

*After-history.*—Not to be traced.

CASE 46.—C. P., æt. 46. Dr. Galabin, Dorcas 7. March 6th, 1894. Married, ten children, three miscarriages. The last pregnancy three years previously. Admitted for hæmorrhage and pain. Six months before admission a discharge commenced. At first this was yellow, and later became streaked with blood. Later there was hæmorrhage on coitus. Pain had been present for two or three months in hypogastric regions and left thigh.

*On examination*, a projecting growth, bleeding readily, was found.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. No great difficulty. Clamps used for the broad ligaments.

*Parts removed* showed that the growth was three and a-half inches across.

*Progress* uneventful. Recovery.

*After-history.*—Not to be traced.

CASE 47.—R. R., æt. 34. Dr. Galabin, Dorcas 16, July 21st, 1894. Married, seven children. Was pregnant on admission. Admitted for repeated

floodings. Had suffered from great irregularity of menses for two years. Hæmorrhages often three times in a month. Two months before admission had hæmorrhage on coitus and constant discharge like "green waters." Severe hæmorrhage occurred nearly every day during this latter period. Micturition painful.

*On examination*, was found to be three or four months pregnant, and had a large cauliflower growth projecting from the cervix, which bled readily on touching. Patient aborted soon after admission, probably owing to vaginal plugging. Afterwards had some septic disturbance, which disappeared in a few days.

*Operation*.—Supra-vaginal amputation. Ligatures used for the bases of the broad ligaments

*Progress* uneventful. Recovery.

*After-history*.—Lived five years after the operation, and died of recurrence.

CASE 48.—M. W., æt. (?). Dr. Galabin, Dorcas 9, February 19th, 1894. Married, six children, one miscarriage. The last pregnancy four months previously. Admitted for incontinence of urine. Four months previously miscarried at two and a-half months. Since then had constant trickling away of urine. Some slight yellow discharge was present, and some back-ache.

*On examination*, a large mushroom-shaped growth was found, with a central depression indicating the position of the external os uteri. Some fixation of the uterus was noted.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Vaginal hysterectomy after first removal of the projecting growth with the *ecraseur*. There was also a hydrosalpinx on the left side, which was removed.

*Parts removed* showed the growth to be a hard carcinoma.

*Progress* uneventful. Recovery.

*After-history*.—Not to be traced.

CASE 49.—C.D. æt. 51. Dr. Horrocks, Dorcas 22, July 9th, 1894. Admitted for pain. Married, three children, the last thirteen years previously. Menopause at 49. The growth on the cervix was found out accidentally when in hospital for piles. Had had some slight discharge but no hæmorrhage. Had had pain at coitus. Some pain and difficulty of micturition.

*On examination*, cervix enlarged by growth, which reached nearly to posterior vaginal reflexion and extended more to the left side than the right; no bleeding on examination. A small ulcerated spot was found.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Supra-vaginal amputation. Ligatures used to control vessels in the broad ligaments.

*Progress*.—Uneventful, except that patient seems to have had pain of some kind since the operation. Recovery.

*After-history*.—Growth recurred and the patient died in March, 1895.

CASE 50.—S. E. æt. 61. Dr. Galabin, Dorcas 7, October 7th, 1895. Married, twelve children, two miscarriages. The last pregnancy 16 years previously. Menopause 15 years previously. Six weeks before admission there was some

hæmorrhage lasting eighteen days, followed by a yellow foul discharge. Some pain in the inguinal regions.

*On examination*, a projecting papillary growth found springing from posterior lip of the os uteri; its base reached the vaginal reflection posteriorly. There was some bleeding on manipulation.

*Operation*.—Vaginal hysterectomy. No difficulty. Clamps and ligatures used for the broad ligaments.

*Parts removed* showed that both lips of the os uteri were involved and that the growth was spreading somewhat upwards.

*Progress*.—Uneventful. Recovery.

*After history*.—Not to be traced.

CASE 51.—T. G. æt. 44. Dr. Galabin, Dorcas 13, February 20th, 1895. Married, seven children, one miscarriage. The last pregnancy nine years previously. Admitted for watery discharge. Symptoms began eighteen months before admission, with a slight blood stained discharge between the menstrual periods, which were normal. Later there was hæmorrhage on coitus, and then the discharge became watery and sometimes foul. There had been no pain and very little wasting; no micturition troubles.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Vaginal hysterectomy.

*Parts removed*.—Not described in the report.

*Progress*.—Only interrupted by some slight pyrexia. Recovery.

*After-history*.—Not to be traced.

CASE 52.—J. H., æt. 43. Dr. Galabin, Dorcas 17, September 30th, 1895. Married, six children, three miscarriages, the last pregnancy seventeen and a half years previously. Menstrual periods had been rather frequent for many years, viz., every three weeks, but only lasted three days. Four months before admission a vaginal discharge, which had been present fifteen years, became much more copious, yellow in colour and streaked with blood. Pain in the back was present and there was considerable wasting.

*On examination*, the posterior lip of the cervix presented a thickened appearance, ulcerated on the surface. No infiltration of vaginal walls. Some bleeding on examination.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Vaginal hysterectomy, by clamps and ligatures. Difficult owing to the narrowness of the vagina.

*Parts removed* showed the growth, as stated above, with very little invasion of the uterus. Microscopically the growth was a mixed cell sarcoma.

*Progress* uneventful. Recovery.

*After-history*.—Was seen in the summer of 1897 and shewed no signs whatever of recurrence. May, 1901, no recurrence, and is quite well.

CASE 53.—L. L., æt. 58. Dr. Galabin, Dorcas 14, July 31st, 1895. Married, seven children, several miscarriages, the last pregnancy eighteen years previously. Admitted for foul sanious discharge. Menopause, eight years before admission. Six months before admission began to lose a blood stained discharge, which later increased in quantity and became foul. No pain. No wasting. No micturition troubles.

*On examination*, the whole os uteri was occupied by an ulcerated freely bleeding growth, which extended to the vaginal reflection posteriorly, and seemed more marked on the left side.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Supra-vaginal amputation. Bleeding arrested by pressure forceps.

*Parts removed* showed that all the growth had been removed.

*Progress* uneventful. Recovery.

*After-history*.—Cannot be traced.

CASE 54.—M. A. L., æt. 42. Dr. Galabin, Dorcas 9, January 26th, 1895. Married, one child, one miscarriage. The last pregnancy fifteen years previously. Menstruation had occurred every fourteen days for the last twelve months attended by much clotting and profuse loss. Between the periods there was a discharge which during the nine weeks before admission became watery and very offensive. Pain had been more or less present for eight months. Bleeding on coitus, and frequent painful micturition had been present during this time.

*On examination*, a mushroom shaped growth confined to the cervix, with an ulcerated surface and everted edges. Bleeding on manipulation.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Supra-vaginal amputation. Some difficulty in stripping the bladder, which came very close to the growth.

*Parts removed* shewed that the growth had been completely removed as far as the naked eye could tell.

*Progress* was interrupted by the formation of an abscess in the cellular tissue to the right of the uterus and posteriorly. This burst into the vagina eventually. Recovery.

*After-history*.—Died from a recurrence of growth in March, 1897.

CASE 55.—E. T., æt. 33. Dr. Horrocks, Dorcas 19, February 3rd, 1895. Married, one child, nine years previously. Admitted for hæmorrhage following coitus. This hæmorrhage occurred three months before admission. Beyond some chronic leucorrhœa there were no other symptoms.

*On examination*, the cervix was found to bleed easily, but was not ulcerated. A small piece removed for examination proved microscopically to be carcinoma.

*Operation*.—Supra-vaginal amputation of the cervix. No difficulty. Hæmostasis with six Spencer Wells' forceps.

*Parts removed* shewed that the growth was of small extent, and that healthy cervix had been cut away an inch above the growth.

*Progress of the case* was uneventful. The forceps were removed on the third day. Recovery.

*After-history*.—Cannot be traced.

CASE 56.—E. W., æt. 57. Dr. Horrocks, Dorcas 23, June 27th, 1895. Married, six children, one miscarriage. The last pregnancy twenty-three years previously. Admitted for hæmorrhage. Menopause occurred at 53. Bleeding commenced again eight months before admission. No other discharge noted. No pain or wasting until about a month before admission. No condition on admission noted; but it must have been a very extensive growth.

*Operation* was commenced as an amputation of the cervix, but finding that the growth went higher than the incision, and also that there were suppurating cavities in the uterine muscle, it was finished as a vaginal hysterectomy. The lower parts of the broad ligament were clamped, the upper parts tied with silk ligatures.

*Parts removed* showed most extensive infiltration of the uterus, with growth.

*Progress of the case.*—The temperature, which had been of a hectic type before the operation, rose continuously afterwards until the patient's death, on the third day, from peritonitis.

*Autopsy.*—General peritonitis found. No hæmorrhage. No growth left behind. No doubt was infected from the pus cavities in the uterine wall.

CASE 57.—M. H., æt. 40. Dr. Galabin, Dorcas 12, April 24th, 1896. Married, three children, one miscarriage. The last pregnancy eleven years previously. During five months before admission there had been hæmorrhages every two or three days, with a white discharge constantly present. Once the hæmorrhage had been profuse. No pain, wasting, or micturition troubles during this period.

*On examination.*—The posterior lip of the cervix was found to be widened and infiltrated by growth. The posterior vaginal wall seemed somewhat affected. The growth bled readily on manipulation.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Clamps used for the broad ligaments.

*Parts removed* not described.

*Progress.*—Interrupted by an attack of tonsillitis. Recovery.

*After-history.*—The growth recurred, and death took place in August, 1900.

CASE 58.—E. H., æt. 54. Dr. Galabin, Dorcas 15, October 15th, 1896. Married. Last pregnancy thirteen and a half years previously. Admitted for continuous hæmorrhage. Menopause occurred ten years previously. Six months before admission hæmorrhage commenced, and had continued. There had been wasting and pain. No micturition troubles.

*On examination.*—The cervix was indurated and bled freely when touched. The cervix was found to be fixed, having a very small range of movement in any direction. Broad ligaments apparently free from growth.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy.

*Parts removed* were not described.

*Progress* uneventful. Recovery.

*After-history.*—The growth recurred, and the patient died in August, 1897.

CASE 59.—S. K., æt. 53. Dr. Galabin, Dorcas 16, October 23rd, 1896. Married, eleven children, three miscarriages. The last pregnancy fifteen years previously. Menopause three years previously. Admitted for a watery discharge, with occasional hæmorrhages, which had persisted for three and a half months before admission. No pain, no wasting, no micturition troubles.

*On examination.*—The finger entered a ragged cavity through the external os. The edges of os uteri and surface of growth inside were ulcerated, and bled freely on touching. No loss of mobility of the cervix.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. No difficulty. Clamps and ligatures were used for the broad ligaments

*Parts removed* showed that all incisions were wide of the growth.

*Progress* uninterrupted, except for constipation. Recovery.

*After-history.*—The patient died in March, 1898, no doubt from a recurrence of the growth.

CASE 60.—C. M. æt. 42. Dr. Galabin, Dorcas 20, November 5th, 1896. Married, nine children, one miscarriage. The last pregnancy was six years previously. Patient had suffered from menorrhagia for about twelve months, and for a little over a month before admission she had profuse irregular bleedings. There was pain on micturition and constant aching pain, worse at night.

*On examination.*—The whole cervix was infiltrated by growth, especially anteriorly. There was a nodule of growth on the anterior vaginal wall. Free bleeding on examination.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Clamps and ligatures were used for the broad ligaments.

*Parts removed* showed that the growth extended up to the internal os and appeared to be all removed.

*Progress* was interrupted by some pyrexia. Recovery.

*After-history* cannot be traced.

CASE 61.—F. P., æt. 59. Dr. Galabin, Dorcas 7, March 24th, 1896. Married, four children, the last pregnancy twenty-one years previously. Menopause occurred fourteen years previously. For three or four years had suffered with prolapse of the uterus. A month before admission a watery discharge began. No bleeding, no pain, no wasting, no micturition troubles.

*On examination.*—A mushroom-shaped growth was found, most marked anteriorly and on the right side spreading on to the vagina.

*Diagnosis.*—Carcinoma of cervix in a prolapsed uterus.

*Operation.*—Vaginal hysterectomy. Considerable difficulty in separating the bladder. Clamps and ligatures used for the broad ligaments.

*Parts removed* showed the growth to be a soft one, and that it was very close to the bladder if it had not actually infiltrated it.

*Progress* uneventful. Recovery.

*After-history.*—Cannot be traced.

CASE 62.—H. P., æt. 38. Dr. Galabin, Dorcas , April 11th 1896. Married, one child, sixteen years previous to admission. Very doubtful how long the history of this case really was. There was menorrhagia, hæmorrhage on coitus, pain and wasting.

*On examination.*—The anterior lip of the cervix was indurated and ulcerated and bled on examination. A piece scooped out of this proved microscopically to be a glandular carcinoma.

*Operation.*—Vaginal hysterectomy. Bladder very difficult to separate and rectum was accidentally opened. This latter was sewn up at once. Clamps used for the broad ligaments

*Parts removed* showed that the growth extended only about half an inch up the cervix, and seemed to have been all removed.

*Progress* uneventful. *Recovery*.

*After-history*.—May, 1901, is reported to have no recurrence of growth, and had just recovered from an operation for floating kidney.

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CASE 63.—F. B., æt. 30. Dr. Galabin, Dorcas 9, December 9th, 1896. Married, five children, the last three years previously. Menstruation had not been regular since the birth of the last child. There had been no excessive hæmorrhage but for three months before admission there had been a watery discharge.

*On examination*.—The vagina was filled with a cauliflower mass, springing from the cervix. This bled readily and obscured the os uteri.

*Diagnosis*.—Carcinoma of cervix.

*Operation*.—Vaginal hysterectomy. Clamps were used for the broad ligaments.

*Progress* uninterrupted. *Recovery*.

*After-history*.—Was seen on February 9th, 1897, and showed no sign of recurrence. However, she died in July, 1897, probably of a recurrence of growth.

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CASE 64.—A. S., æt. 57. Dr. Horrocks, Dorcas 9, March 31st, 1896. Married, ten children, one miscarriage. The last pregnancy eighteen years previously. Admitted for hæmorrhage. Menopause occurred at 52. Hæmorrhage commenced again nine months before admission. Was said to have had two polypi removed two months before admission. No note as to pain or wasting.

*On examination*.—Uterus is found to be only slightly enlarged, bleeds freely on passing sound. Pieces scraped away with a dull curette and examined microscopically, showed carcinomatous structure.

*Operation*.—Vaginal hysterectomy. Clamps and ligatures used for the broad ligaments.

*Parts removed* shewed two small nodules at fundus looking like small fibroids with breaking down ulcerated carcinomatous growth over them.

*Progress of the case* was complicated by some suppuration at the top of the vagina; however the temperature never rose above 100·6°, and all symptoms subsided when the ligatures were removed on the twenty-eighth day. *Recovery*.

*After-history*.—Reports herself in May, 1901, as quite well.

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CASE 65.—M. A. W., æt. 55. Dr. Horrocks, Dorcas 21, January 16th, 1896. Married, seven children, the last twenty years previously. Admitted for hæmorrhage and pain. The menstrual periods had remained regular up to three years before admission. They recurred every four weeks and were attended by very copious flow, and often by the passage of clots. Two and a half years before admission had a free hæmorrhage, and passed a mass which was supposed to be a polypus. Bleeding ceased for eighteen months, and then eleven months before admission came on again and continued. There was at this period pain in the lower part of the stomach.

*On examination*, a swelling, the size of a walnut, could be felt projecting from the cervix. This was continuous with a large mass in the uterine wall, on the left side.

*Diagnosis.*—Fibro-myoma of uterus.

*Operation.*—The whole mass was removed by morcellation, and the cavity packed.

*Progress* was uneventful and the patient went out apparently well.

*After-history.*—Was readmitted on October 11th, 1897, for discharge and loss of flesh. A large tumour could be felt on the right side above Poupart's ligament, and per vaginam, a mass projecting through the dilated cervix. There had been some difficulty in holding urine occasionally. The growth was still thought to be a fibroid, and was partly enucleated and partly removed by morcellation; seventeen ounces of growth were thus removed, it was not examined microscopically. Patient again did well.

*Readmitted* on June 27th, 1897. Has had a continuous discharge since last operation. The fundus uteri now reaches to the level of the anterior superior spine on the left side. Some of the tumour was again removed by morcellation and enucleation. Much, however, was left. It was examined microscopically and was found to be a spindle-celled sarcoma. July 10th. Still a large abdominal tumour, and again a piece fungating into vagina. Some more removed. Patient very emaciated and not expected to survive much longer. Further history not to be traced.

CASE 66.—K. C., æt. 36. Dr. Galabin, Dorcas 22, April 22nd, 1897. Married, six children, one miscarriage. The last pregnancy was three years previous to admission. For some months previous to admission there had been menorrhagia, gradually getting worse. Advice had only been sought three months before admission. There had been no pain, but patient was very anæmic, with a feeble, rapid pulse, and had some wasting.

*On examination.*—Showed the cervix to be deeply cleft on each side, the two lips being hypertrophied and nodular. A piece removed with the curette proved microscopically to be glandular carcinoma.

*Operation.*—Vaginal hysterectomy, with clamps and ligatures for the broad ligaments.

*Parts removed* showed that the growth extended up the canal of the uterus to within an inch of the fundus.

*Progress* was unsatisfactory. The patient was very blanched after the operation, and died on the third day. There apparently was no sign of septicæmia or peritonitis.

*Autopsy* showed that death was due to septicæmia from gangrene of the left lung. There was no peritonitis nor hæmorrhage.

CASE 67.—M. C., æt. 39. Dr. Galabin, Dorcas 18, November 26th, 1897. Married, nine children, two miscarriages. The last pregnancy two and a half years previously. Eight months before admission the periods became excessive, and there were occasional intermenstrual losses. Later there was a constant foul discharge. Backache was present. No wasting; no micturition troubles.

*On examination.*—The whole cervix was found to be excavated by a soft growth, which bled very freely on touching.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Clamps and ligatures used. Much difficulty in separating bladder.



*Parts removed* showed that there was very little tissue between the growth and the bladder, and it was doubtful if all growth was removed. The growth had begun within the cervix.

*Progress* uneventful. *Recovery.*

*After-history.*—February 28th, 1898, was examined, and there was undoubted evidence of recurrence in the scar at the top of the vagina. Further history cannot be traced.

CASE 68.—A. D., æt 47. Dr. Galabin, Dorcas , September 28th, 1897. Married, one child, two miscarriages, the last pregnancy twenty years previously. Three months before admission a watery discharge commenced, which gradually became very foul. The menstrual periods were quite regular and distinct though somewhat increased in quantity of blood lost. There was no pain, wasting nor micturition trouble.

*On examination.*—A flat growth commencing in the anterior lip and spreading towards the bladder was found. Microscopically it was a columnar celled carcinoma.

*Operation.*—Supra-vaginal amputation.

*Progress* uneventful. *Recovery.*

*After-history.*—June, 1898: No evidence of recurrence. Had much dysmenorrhœa of a spasmodic nature and evidently due to cicatrization and obstruction following on the operation. December, 1899:—Pan hysterectomy, by the abdominal method, was performed for the relief of the dysmenorrhœa. There was no evidence of recurrence, but there were several small fibroids in the wall of the uterus. *Recovery.* May, 1901: Reports herself in good health.

CASE 69.—E. H., æt. 52. Dr. Galabin, Dorcas 7, January 3rd, 1897. Married, six children, one miscarriage. The last pregnancy seventeen years previously. Menstruation was regular until two month before admission, when the last two periods were prolonged and the loss was excessive. Had suffered from cervical erosion eighteen months before admission. During the six weeks before admission had a constant blood-stained watery discharge. There had been pain referred to the vagina, wasting and frequent micturition.

*On examination.*—The prominence of the cervix was seen to be replaced by an ulcerated surface. The mobility of the uterus was diminished and the left utero-sacral ligament seemed contracted.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Clamps and ligatures used.

*Parts removed* showed that the right broad ligament was probably beginning to be infiltrated by growth. It was very doubtful if all the growth was removed.

*Progress* was fairly satisfactory. *Recovery.*

*After-history.*—Growth recurred, and the patient died in August, 1898.

CASE 70.—A. H., æt. 50. Dr. Horrocks, Dorcas , April 5th, 1897. Married, three children, the last twenty-two years previously. Admitted for hæmorrhage and pain. Menopause occurred three years before and from that time until nine months before admission patient had no discharge. Then a watery discharge commenced, attended with pain in abdomen and sacrum.

This was never blood-stained until three weeks before admission, when hæmorrhage started.

*Diagnosis.*—Growth of cervix.

*Operation.*—Vaginal hysterectomy. No difficulty. Hæmostasis by Spencer Wells' forceps.

*Parts removed.*—No description.

*Progress of the case* uneventful except for a fixed pain referred to the left lumbar region. Recovery.

*After-history.*—May, 1901, is reported to have a recurrence in the scar since May, 1900. Is not yet very bad and suffers no pain.

CASE 71.—S. L., æt. 42. Dr. Horrocks, Dorcas 23, August 17th, 1897. Married, eight children, the last five and a half years previously. Admitted for continuous hæmorrhage. Menstrual periods have been prolonged during the eight weeks preceding admission. Three months before admission a discharge of yellowish colour commenced and then the above-mentioned menstrual disturbance. There has been no local pain, but some in left side for six months. No micturition troubles.

*On examination.*—A large cauliflower growth involving both lips of cervix and extending on to posterior vaginal wall. Very friable and bleeds readily.

*Diagnosis.*—Epithelioma of cervix.

*Operation.*—Supra-vaginal amputation. There was no possibility of cutting beyond the growth posteriorly, on account of the danger of opening the rectum. Spencer Wells' forceps applied to the broad ligaments.

*Parts removed* consisted of cervix and portions of growth.

*Progress of the case* was interrupted owing to a profuse secondary hæmorrhage, which was controlled by plugging on the eighth day. Recovery.

*After-history* cannot be traced.

CASE 72.—E. P., æt. 45. Dr. Horrocks, Dorcas 11, June 24th, 1897. Married, eleven children, the last three years previously. Admitted for hæmorrhage and pain. Monthly periods had been regular until ten months before admission when she had a flooding. Then a watery discharge commenced and was accompanied by hypogastric pain.

*On examination.*—The uterus was enlarged and a cauliflower mass projected from the cervix. The cervix itself seemed very hard.

*Operation.*—Vaginal hysterectomy, rather difficult owing to the large size of the uterus. Spencer Wells' forceps applied to the broad ligaments.

*Parts removed* showed that the cervix was infiltrated with growth as far as the internal os. The growth had invaded the peritoneum of Douglas' pouch. The body or the uterus was not dilated, but the walls were thick and œdematous.

*Progress of the case* was uneventful. Recovery.

*After-history.*—Growth recurred, and the patient died in April, 1898.

CASE 73.—E. W., æt. 62. Dr. Horrocks, Dorcas 20, March 22nd, 1897. Married, no children. Admitted for pain and hæmorrhage. Menopause at 50 years. Hæmorrhage started twelve months before admission. This continued with intermissions of two to three weeks at a time. Some dragging pain in the right side. Occasionally difficulty in micturition. Some wasting.

*On examination.*—The cervix was very hard and bled on examination. There was a scar from an old tear running to the left vaginal wall. No ulceration detected.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Spencer Wells' forceps applied to broad ligaments.

*Parts removed* shewed the entire cervix infiltrated with growth up to the internal os. Uterine body very small.

*Progress of case* uneventful. Recovery.

*After-history.*—The patient was better for six months after the operation, but gradually became worse and died in June, 1900.

CASE 74.—R. B., æt. 54. Dr. Galabin, Dorcas 20, February 24th, 1898. Admitted for pain in the back and irregular hæmorrhages. Married, six children. Four months previously the pain commenced with backache. A month later the first hæmorrhage occurred for no particular reason. The hæmorrhages recurred at intervals, and latterly occurred during coitus. There was pain and difficulty in micturition.

*Per vaginam.*—An ulcerated growth with everted edges on anterior lip of cervix, extending up to the vaginal reflexion. Uterus only fairly mobile. Foul discharge in the vagina.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Supra-vaginal amputation of cervix. Much difficulty in separating the bladder. Clamps applied to lower parts of broad ligaments. Peritoneum not opened.

*Parts removed*, consisted of cervix infiltrated with soft friable growth everywhere except for a small area posteriorly. Only a thin shell of tissue left where the bladder had been dissected off. Normal uterine tissue above the growth.

*Progress of the case* was only interrupted on the sixth day by a rigor and rise of temperature which lasted four days. Recovery.

*After-history.*—The growth recurred, and the patient died in January, 1899.

CASE 75.—J. B., æt. 47. Dr. Galabin, Dorcas, May 23rd, 1898. Admitted for red discharge and pain in the hypogastric region. Widow, two children. Had been operated on eleven years previously for "matted appendages," and also two years previously for a ventral hernia. The red discharge began seven months before admission, there having been no discharge of any kind since the first operation until then. Marked wasting for four months before admission. No micturition troubles.

*Per vaginam.*—The external os and cervix are found to be replaced by a conical ulcer with hard everted edges. The ulcer extends on to the anterior and left lateral vaginal walls. Mobility of uterus good.

*Diagnosis.*—Carcinoma of cervix uteri.

*Operation.*—Vaginal hysterectomy. Much difficulty in separating bladder from the growth. Lower half of broad ligaments clamped, upper halves ligatured.

*Parts removed.*—The cervix was completely infiltrated with a warty new growth all round. The growth extended upwards above the internal os. The growth in front was very close to the bladder, but it had not actually infiltrated it. The body of the uterus was very small and atrophied.

*Progress.*—Clamps removed in forty-eight hours. Ligatures came away on the twelfth day. Recovery uninterrupted.

*After-history.*—Came to Surrey Dispensary to see the author, 23rd February, 1901. Looked fat and well. No sign of recurrence whatever.

CASE 76.—A. E., æt. 38. Dr. Galabin, Dorcas 19, March 28th, 1898. Married five years, no children, one miscarriage four years ago. Menstruation regular; three weeks' interval, seven days' duration. Admitted for uterine discharge, pain and prolapse. Uterus first prolapsed a year before admission. The first hæmorrhage, apart from menstruation, occurred four months before admission. At that time there was a copious watery discharge, which latterly became blood-stained and offensive. Some difficulty of micturition, due to the prolapse.

*On examination.*—The uterus was found about half outside the vulval orifice. The external os was displaced to the left by a mass of ulcerated growth on the right side of the cervix. This did not bleed; but there was a thin stream of blood issuing from the external os. The whole uterus was painful to the touch, and the sound passed four and a half inches.

*Diagnosis.*—Carcinoma of cervix in a prolapsed uterus.

*Operation.*—Vaginal hysterectomy. Some difficulty with bladder, so that at one point only the mucous membrane was left when stripped off. Here a suture was inserted to strengthen the bladder wall. Broad ligaments clamped below; ligatured above.

*Parts removed* showed that the growth had infiltrated the whole circumference of the cervix, except a small area behind and to the left. The growth was covered by cervical mucous membrane internally, and by vaginal mucous membrane, except at the most dependent part, which was ulcerated and sloughy. The bladder had been attached over an area which was two and three-quarter inches from above downwards, accounting for the difficulty in separating it.

*Progress* was uninterrupted, except for a mild attack of cystitis. The clamps were removed in forty-eight hours without hæmorrhage. The sutures came away before her discharge on the twenty-fourth day. Recovery.

*After-history.*—Came up on June 6th, six weeks later, with a mass of granulations in the right fornix, which looked like a recurrence. These were removed, and the base cauterised. Further history not to be traced.

CASE 77.—S. G., æt. 40. Dr. Galabin, Dorcas 16, July 15th, 1898. Admitted for a tumour and pain in the back and abdomen. Married, ten children; the last born November, 1896. In November, 1897, the patient had a sudden flooding, which continued more or less until March, 1898. In February, the growth had been diagnosed by a doctor. Pain commenced in April, 1898. She had lost much flesh, and had had difficulty in passing urine.

*On examination.*—The cervix was found to be expanded by a growth, which fungated through the external os somewhat.

*Diagnosis.*—Glandular carcinoma, starting in the cervical canal.

*Operation.*—Vaginal hysterectomy, with clamps for both lower and upper parts of the broad ligaments. No particular difficulty in the operation.

*The parts removed* showed that the whole cervix was expanded and infiltrated by a very hard growth, which measured two and a half inches in diameter,

and extended up into the body of the uterus two and a-half inches from the lowermost point.

*Progress of the case* was unsatisfactory, as some days after the clamps were removed, a vesico-vaginal fistula was found, which it was deemed impossible to close. Recovery, with vesico-vaginal fistula.

*After-history.*—Recurrence took place, and the patient died in December, 1898.

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CASE 78.—S. L., æt. 33. Dr. Galabin, Dorcas 21, September 10th, 1898. Admitted for thirteen weeks' continuous hæmorrhage. Married, seven children. The last child was born twenty months before admission. No menstruation since last confinement. Thirteen weeks before admission the hæmorrhage began, and continued more or less. There was no pain nor wasting. No difficulty of micturition.

*On examination.*—She was found to be five months pregnant, and in addition had a growth in the cervix. The whole cervix seemed to be involved, and was about the size of a five shilling piece in diameter. Induction of labour was performed, and the fœtus removed with some difficulty. A month later, the patient having in the meantime been to a convalescent home, she was readmitted for operation. The growth now seemed smaller, and was in a much more healthy condition, as a result of repeated antiseptic douching; but it had invaded the vaginal vault on the left side. The uterus seemed freely movable, and there was no evidence of peritoneal infiltration.

*Operation.*—Vaginal hysterectomy. A good deal of difficulty was experienced in separating the bladder. Clamps were used for the lower parts of the broad ligaments, No. 4 silk ligatures for the upper parts.

*Parts removed.*—Uterus and cervix infiltrated by growth.

*Progress of the case* was uneventful. The clamps were removed in forty-eight hours; no hæmorrhage. The ligatures came away on the fifteenth day. Recovery.

*After-history.*—In September, 1900, two years after the operation, she came up to see Mr. Targett, who reports that patient was looking very ill and anæmic. Has much foul discharge, but not much pain. The growth had recurred, and extended down the vaginal wall to within one inch of the outlet. In May, 1901, she is reported to be dying of the recurrent growth.

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CASE 79.—F. M., æt. 36. Dr. Galabin, Dorcas 18, June 5th, 1898. Admitted for irregular hæmorrhage and pain. Married, no children. Menstruation regular until six months previous to admission, when the loss was every fortnight. Much leucorrhœa for a year. Six weeks before admission had a flooding, and had been losing more or less ever since. During this six weeks she had pain, mostly in the groins. Frequent micturition, every hour almost, had been the rule of late. Wasting had occurred during this time. The growth could be just felt at the external os, and seemed to affect the whole circumference of the cervix. It was rather soft and friable, and commenced some distance in the cervical canal.

*Diagnosis.*—Glandular carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. No difficulty at all. Clamps used for lower halves of broad ligaments, silk ligatures for the upper halves.

*Parts removed* showed that the whole circumference of the cervix was infiltrated with a soft carcinoma, so that only a thin shell of tissue remained

posteriorly. There was more uterine tissue unaffected in front. There was a tag of omentum adherent to the fundus, which had torn through during the operation. Microscopically the growth was a soft glandular carcinoma.

*Progress of the case* uneventful. Ligatures came away by the twentieth day. *Recovery.* Left hospital on July 2nd, 1898.

*After-history.*—Came back on October 3rd, 1898, with a recurrent growth at the top of the vagina. This was cut out with scissors and the cavity burnt with Paquelin's cautery. March 12th, 1899: complains of pain in right hip; has a recurrent growth chiefly in the right broad ligament. No flooding since the operation in October, 1898. December 27th, 1900: Growth has now spread to the bladder and there is a vesico-vaginal fistula high up. The growth has spread down the vaginal walls to the orifice. Blood-stained discharge for a year past. Very thin and ill-looking, and cannot sleep on account of pain in the hips and thighs.

**CASE 80.**—A. P., æt. 41. Dr. Galabin, Dorcas 17, May 2nd, 1898. Married, nine children. Menstruation normal until November 1st, 1897, when she began to lose irregularly and did so until her admission. Since February, 1898, there had been a continuous brown foul discharge. There had been no pain, very little wasting, no trouble with micturition.

*On examination.*—The external os uteri was found to be patent and the interior of the canal was hard, nodular and irregularly excavated. The fornices were free and the uterus quite movable.

*Diagnosis.*—Carcinoma starting in the cervical canal.

*Operation.*—Vaginal hysterectomy. No difficulty during operation. Clamps used for lower half of broad ligaments, ligatures for upper halves.

*Parts removed* showed a very much enlarged cervix infiltrated uniformly with growth. The walls of the cervix were half an inch thick, composed of firm white growth with a very thin shell of uterine tissue on the anterior aspect and very little above posteriorly. The interior of the cervix was an irregular rather wide ulcerated cavity. The body of the uterus was hypertrophied, but the cavity not dilated. The whole uterus measured five inches in length of which nearly three inches was cervix.

*Progress of the case.*—From the first there was some rise in temperature, which continued for a fortnight, the highest recorded temperature being 103.2°. The clamps were removed in forty-eight hours without hæmorrhage. No note as to the sutures. Eventually the temperature came down. *Recovery.*—Went out on June 4th, 1898.

*After-history.*—July 4th some thin purulent discharge complained of, which seemed to be caused by some granulations at the top of the vagina. These were treated with nitrate of silver. No sign of recurrence. May, 1899: A year after operation, patient quite well, no sign of recurrence. The growth recurred later and death occurred from it in February, 1901.

**CASE 81.**—C. W., æt. 53. Dr. Galabin, Dorcas, November 29th, 1898. Admitted for vaginal discharge. Married, seven children, the last twenty years ago. Menopause at 40. Seven months before admission had a "muddy" discharge and some pain in the abdomen. Four months before admission the discharge was always streaked with blood. No floodings. No micturition nor defæcation troubles. Much loss of flesh.

*On examination.*—The cervix was thickened and indurated, the os was patent and through it a hard granular surface within could be felt. Per rectum there seemed to be some thickening of the right utero-sacral fold. No bleeding on examination.

*Diagnosis.*—Carcinoma starting in cervical canal.

*Operation.*—Was commenced in the usual way for vaginal hysterectomy, but after clamping the lower parts of the broad ligaments it was found that the uterus could not be anteverted or retroverted to ligature the upper parts of the ligaments. Consequently the abdomen was opened and the operation completed from above. The difficulty of the case lay in several factors; first the small size of the vagina, atrophied by the premature menopause; second, the large size of the fundus; third, the rotten state of the cervix, which would not hold tenacula.

*Parts removed* showed that a most extensive growth had infiltrated the whole cervix and the lower two-thirds of the body of the uterus. The growth had also reached the peritoneum of Douglas' pouch and gone some distance into the broad ligaments. The disease was really too far advanced for operation. Microscopically the growth was a glandular-celled carcinoma, starting in the cervical mucous membrane.

*Progress of the case.*—The patient was very collapsed after the operation, which lasted nearly three hours, and in the evening of the same day four pints of saline solution were infused into the brachial vein. Next day the patient was vomiting, could not retain enemata, had a very weak pulse, and the edges of the abdominal wound looked as if becoming gangrenous. In the evening she became more collapsed and died.

No autopsy was allowed, but the cause of death was probably acute septicæmia. The pulse, which was 120 after the operation, steadily rose to 164 before death.

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CASE 82.—S. F., æt. 63. Dr. Galabin, Dorcas 13, May 10th, 1891. Married, eleven children, the last twenty-four years ago. Menopause at 48. Was admitted for prolapse of the uterus and hæmorrhage. The prolapse seemed to have existed some time, and had been treated with a hard ring pessary. four months before admission there was blood in the discharge for the first time, the discharge having been white previously for two years. Had lost blood continuously more or less for four months. Pain in the left side and groin for a like period. No micturition trouble.

*On examination.*—A hard ulcerated growth could be felt and seen in the posterior lip of the cervix involving the vaginal wall somewhat.

*Diagnosis.*—Epithelioma of cervix.

*Operation.* Vaginal hysterectomy. No difficulty; clamps for broad ligaments below, ligatures above.

*Parts removed* showed an atrophied uterus measuring barely three inches. The growth had started in the posterior lip of the cervix, and had involved the vaginal wall and the cellular tissue just above and to the left. The incision had gone very close to the growth in the left broad ligament. Microscopically it was a squamous-celled epithelioma.

*Progress of the case* was uneventful. The ligatures came away on the sixteenth day. Recovery. Went out on June 20th 1898.

*After-history.*—Came on November 17th 1898, looked very well, no evidence of recurrence. Some trouble from prolapse of the vaginal walls. September

13th, 1900: No recurrence. May, 1901: Reports herself in not very good health, but apparently has no recurrence of the growth.

**CASE 83.**—M. H., æt. 52. Dr. Galabin, Dorcas, December 27th, 1897. Admitted for a watery blood-stained discharge. Married, nine children, three stillborn and two miscarriages. Last pregnancy thirteen years before. Menstruation regular every twenty-eight days. Loses a good amount. The menorrhagia commenced three years previously, and between the losses there was a discharge which in the last few months was watery in character. No pain, except on defæcation. Frequent micturition just previous to admission.

*On examination.*—A cauliflower-like mass of growth was found projecting from the posterior and left edge of the cervix. Microscopically this was a squamous-celled epithelioma (after removal with the écraseur).

*Diagnosis.*—Squamous epithelioma of cervix.

*Operation.*—Projecting growth first removed then vaginal hysterectomy. There was some considerable difficulty in opening the peritoneum in front, because its reflection had been pushed up by a fibroid one and one-twelfth of an inch in diameter in the anterior wall. Clamps were used for the broad ligaments below, ligatures above.

*The parts removed* showed that the growth had infiltrated the posterior lip of the cervix, and had just begun to invade the vaginal mucous membrane. The cauliflower mass measured three inches by two inches in diameter.

*Progress of the case.*—Three weeks after the operation it is noted that the patient has had incontinence of urine for fourteen days, and for a long time it could not be determined whether the fistula was vesical or ureteric. However, two months after the operation a small vesico-vaginal fistula was discovered in the left posterior corner of the vaginal roof, by injecting boiled milk and water. No treatment was directed towards closing this. The ligatures did not come away until five weeks after the operation. Recovery, with vesico-vaginal fistula. Went out on February 27th, 1898.

*After-history.*—May 10th, 1901, reports herself as fairly well, and having no return of her former complaint.

**CASE 84.**—L. D., æt. 48. Dr. Galabin, Dorcas, April 22nd, 1898. Married, nine children; the last eight years ago. Admitted for hæmorrhages following on the expulsion of a hydatidiform mole. Had been regular until May, 1897. From then until August she had amenorrhœa. From August until November 25th there were irregular hæmorrhages, accompanied by progressive weakness and emaciation. On November 25th a body was passed, which, from the doctor's description, was a hydatidiform mole. After this, irregular bleedings occurred at intervals up to the time of admission. There was no pain, in fact no symptoms beyond anæmia and weakness, consequent on the excessive losses.

*On examination.*—The uterus could be made out to be enlarged; the sound passed three and a half inches, and caused some bleeding. The cervix was dilated, and the finger introduced, felt a growth on the anterior wall projecting into the cavity. A piece of this was removed for microscopic examination, and proved to have the structure of *deciduoma malignum*.

*Operation.*—Vaginal hysterectomy. No difficulty, except on account of the large size of the fundus. Clamps and ligatures were used for the broad ligaments.



*The parts removed* showed the growth as described, including the uterine wall, and a microscopic section through growth and uterine wall proved the diagnosis to be correct.

*Progress of the case.*—The clamps were removed in forty-eight hours. The temperature remained about normal, but the bowels never acted, and the patient died on the fifth day with signs of peritonitis.

*Autopsy.*—There was no general peritonitis, but the pelvic viscera and adjacent coils of intestine were glued together with recent lymph. There were no secondary growths. The cause of death was no doubt septicæmia, on account of the intestinal paresis.

CASE 85.—G. B., æt. 34. Dr. Horrocks, Dorcas 21, March 16th, 1898. Married, two miscarriages, the last in 1888. Admitted for pain and hæmorrhage. Menstruation regular until three months before admission; then a white discharge commenced, which six weeks before admission became blood-stained. She had at this time pain in the right side. During this time the patient lost flesh.

*On examination.*—There was found a prominent growth of the posterior lip of the cervix, not invading the vaginal walls, very friable and bleeding freely on examination.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. No difficulty except for a few adhesions on the posterior uterine wall. Hæmostasis by many pairs of Spencer Wells' forceps.

*Parts removed* showed that the growth had arisen in the glandular tissues just inside the external os. By its growth it had infiltrated the cervix and then ulcerated through. It was a soft glandular carcinoma.

*Progress of the case* was uneventful. Recovery.

*After-history.*—June 21, 1898: Examined and a suspicious nodule found in the cicatrix of the vaginal roof. Further history not to be traced.

CASE 86.—S. B., æt. 43. Dr. Horrocks, Dorcas 23, March 1st, 1898. Married, nine children, the last July, 1897. Had never been well since the last pregnancy. The periods which used to last three days now last eight, and for the past six months there had been a watery brownish discharge and some bleeding on coitus. Had had frequent micturition, often getting up two or three times in the night. There had been some backache, but no loss of flesh.

*On examination.*—An ulcerated growth was found spreading out the posterior lip of the cervix to a diameter of one and a half inches. The uterus was fairly movable and the sound passed three inches.

*Operation.*—Vaginal hysterectomy. No difficulty. Hæmostasis by twenty pairs of Spencer Wells' forceps.

*Parts removed* showed the posterior lip with a warty ulcerated growth; the edges of the ulcer were everted. On section, a nodule one inch in diameter was found in the substance of the posterior lip, which had grown in by a narrow neck from the surface. The growth microscopically was a squamous epithelioma with a few well-formed cell-nests.

*Progress of the case* was uneventful. The forceps were removed after thirty hours. Recovery.

*After-history.*—In May, 1901, is reported in good health.

**CASE 87.**—E. C., æt. 56. Dr. Horrocks, Dorcas 21, May 26th 1898. Married, three children, the last fifteen years ago. Admitted for painful micturition and discharge. Menopause at fifty years. Had been quite well until two years before admission; then she began to have a red discharge, much pain in the lower abdomen and during these two years had four attacks of hæmorrhage. Later the pain was of a neuralgic character and radiated down the right leg. Fourteen days before admission she began to have painful micturition.

*On examination.*—The growth found was very extensive, infiltrating the vaginal walls and bases of the broad ligaments. It was determined not to operate. However, the patient begged to have something done.

*Operation.*—Supra-vaginal amputation. Lower parts of broad ligaments clamped. Patient became very collapsed under anæsthetic and the operation had to be hurriedly terminated.

*Parts removed* consisted of sloughing, horribly foul, masses of growth.

*Progress of the case.*—Patient apparently never rallied, but became progressively weaker and died on the third day after the operation with signs of peritonitis and septicæmia. Death.

*Autopsy.*—The fundus of the uterus, the cut vaginal walls and the bases of the broad ligaments all showed masses of growth. The peritoneum had been opened posteriorly, and showed pus and recent lymph in the pelvis and on omentum. Glands at the pelvic brim were infiltrated with growth.

**CASE 88.**—E. D., æt. 37. Dr. Horrocks, Dorcas, December 5th, 1898. Married, two children, the last twelve years ago. Admitted for discharge and pain. Menstruation had been regular until three or four years before admission. Since then had been irregular, often every fortnight and the losses were profuse. Had suffered from leucorrhœa for years, and during the last two months had noted that the discharge was blood-stained. Had backache some time. No micturition troubles.

*On examination.*—There was found a mushroom shaped tumour the size of a tangerine orange, springing from the anterior part of the cervix. The tumour was not ulcerated and did not bleed readily. There was some fixity of the uterus on the left side.

*Operation.*—Vaginal hysterectomy. The only difficulty experienced was on account of the difficulty of obtaining a good hold. The broad ligaments were clamped with eighteen pairs of Spencer Wells' forceps.

*Parts removed* showed the anterior lip infiltrated by the growth and projecting from it. The endometrium was extremely thick, gelatinous-looking, and rugose. Microscopically the tumour was a glandular carcinoma. The thickened endometrium was of the benign adenomatous type of growth.

*Progress of the case* was uneventful. Recovery.

*After-history.*—Patient was seen on April 18th, 1899. She has had some discharge and occasionally pain in the right hip. A button-like mass, the size of a half-penny, was found at the site of the scar in the vagina, removed with scissors and cauterized with Paquelin's cautery. This was probably a recurrent growth. Died in June, 1900, in St. Mary's Hospital.

**CASE 89.**—F. J., æt. 40. Dr. Horrocks, Dorcas 15, October 31st, 1898. Married, five children, one miscarriage, the last pregnancy October, 1894.

Admitted for constant vaginal discharge. Four months before admission a foul red discharge commenced and had continued ever since. Much pain in back and abdomen. No trouble with micturition. Had lost much flesh.

*On examination.*—A mushroom shaped growth was found projecting from the cervix into the vagina. Uterus and cervix seem quite movable.

*Operation.*—Vaginal hysterectomy. No difficulty, but the anterior pouch of peritoneum was not opened until quite late, and then by depressing the peritoneum with the fingers from above. Fifteen pairs of Spencer Wells' forceps were used to clamp the broad ligaments.

*Parts removed* showed that the growth had invaded the portio vaginalis, but had not reached the body of the uterus. It was a glandular carcinoma microscopically.

*Progress of the case.*—Forceps removed in twenty-eight hours, also gauze plug, which was not replaced. Further progress uneventful. Recovery.

*After-history.*—Cannot be traced.

CASE 90.—H. P., æt. 33. Dr. Horrocks, Dorcas 19, February 18th, 1898. Married, one child ten years previously. Widow. Admitted for discharge and hæmorrhage. Menstruation regular, and for the ten months before admission had a discharge between the periods.

*On examination.*—The interior of the cervix could be felt through the external os to be excavated, and bled on touching.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Considerable difficulty in separating bladder and reaching peritoneum. Eighteen pairs of Spencer Wells' forceps used to clamp the broad ligaments.

*Parts removed* showed that the growth had excavated the cervix up to the internal os, but had not invaded the body of the uterus. In front the growth must have been very close to the base of the bladder. The wall of the uterus was thickened. Glandular carcinoma microscopically.

*Progress of the case.*—Forceps removed in thirty-three hours. Uneventful convalescence. Recovery.

*After-history.*—Readmitted in August, 1898, with a recurrence in the posterior vaginal wall. When cut out this disclosed a further mass of growth towards Douglas' pouch. This was scraped and swabbed with formalin, 1 in 500. October, 1898: Distinct evidence of recurrence, but patient looks well and has no pain. She has, however, an offensive discharge. No further operation possible. Not to be traced further.

CASE 91.—F. L., æt. 53. Dr. Horrocks, Dorcas 23, June 4th, 1898. Widow. No pregnancies. Menopause at 50. One year before admission she had a flooding followed by a red discharge. This continued and she had other floodings. The discharge became offensive three weeks before admission and was accompanied by very frequent micturition. A little aching pain in the back was complained of latterly.

*On examination.*—The sound passed barely three inches, and caused rather free bleeding. The uterus did not seem to be enlarged. A small scraping removed was microscopically found to be carcinomatous. The uterus was freely movable. No evidence of invasion of the bladder.

*Operation.*—Vaginal hysterectomy was commenced but proved very difficult on account of the small vagina and friability of the uterus. When at last the

uterus was nearly free, a large piece from the right corner broke off and was left behind. After some search from below it was decided to open the abdomen from above. On doing this the piece of uterus with growth was found free in the right iliac fossa, quite out of reach from the vagina. No more growth could be found. Spencer Wells' forceps were used to clamp the broad ligaments.

*Parts removed* showed the whole interior of the body infiltrated with growth to within two inches of the external os. Although the sound only passed three inches, the uterus must have been much larger than that. The growth had penetrated right through the peritoneum and had set up adhesions to neighbouring organs, thus helping to make the operation more difficult. Microscopically the growth was a very soft glandular carcinoma.

*Progress of the case* was complicated by septic peritonitis, diarrhoea, bronchitis and sloughing of the abdominal wound. Fortunately the muscle layer held, but the skin separated to four or five inches in the middle. The temperature kept near or subnormal. After a long illness the patient gradually recovered with a sound abdominal scar. Recovery.

*After-history.*—In October, 1899, was seen, and then had no sign of recurrence and the abdominal scar remained sound. In May, 1901, reports herself as quite well.

CASE 92.—H. O., æt. 42. Dr. Galabin, Queen 2, February 18th, 1899. Married, six children; the last six years ago. Admitted for hæmorrhage and pain. Patient was quite well until May, 1898, when she had a flooding between two menstrual periods. After this she had a yellow discharge, which was often blood-stained, and later became very offensive. She had much pain in the left side and lower part of abdomen.

*On examination.*—A large cauliflower growth was found projecting from the cervix and invading the vaginal walls at its base everywhere, except on the anterior wall. The temperature was always raised, between 99° and 100·2°. As a preliminary, this mass of growth was scraped away, all sloughing tissue being thus got rid of.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. No difficulty. Clamps used for lower halves and ligatures for upper halves of broad ligaments.

*Parts removed* showed a carcinoma invading cervix and adjacent parts of vaginal walls, as described above.

*Progress of case.*—The temperature kept up after the operation, and varied between 103° and 98·4°, being evidently of a septicæmic type. The patient went out on the thirteenth day, still with high fever, in spite of the warning given by the physician in attendance.

*After-history.*—Patient never recovered, and died in July, 1899.

CASE 93.—S. R., æt. 47. Dr. Galabin, Queen 20, July 19th, 1899. Married, four children; the last six years ago. Admitted for slight continuous hæmorrhage and pain. Since the last child, six years previously, the patient had only two menstrual periods, but was quite well until three months before admission. Then she had slight continuous hæmorrhage and some backache for six weeks. This hæmorrhagic discharge apparently continued until admission.

*On examination.*—The cervix, which was atrophic, easily admitted the finger, which felt an irregular ulcerated cavity inside. There was no fixation of the uterus. No micturition troubles, and very little discharge.

*Diagnosis.*—Carcinoma, beginning in the cervical canal.

*Operation.*—Vaginal hysterectomy. No difficulty. Clamps and ligatures for the broad ligaments.

*Parts removed* showed that the growth involved the whole of the cervical canal, and extended a little above the external os. It was doubtful whether all the growth had been removed. No dilatation of the uterine cavity.

*Progress* uneventful. Clamps removed in thirty six hours. Ligatures came away on the nineteenth day. Recovery.

*After-history.*—May, 1901. Reports that there is no sign of recurrence.

CASE 94.—L. W., æt. 40. Dr. Galabin, Queen 5, April 20th, 1899. Married, five children; the last five years previously. Admitted for discharge. Five months before admission a discharge commenced, which was alternately pure blood, slightly blood-stained, or like "dirty water." No pain at any time. Menstruation quite regular. Loss of flesh for two months. No micturition troubles.

*On examination.*—A lobulated growth found occupying the cervix, and spreading to vaginal wall posteriorly. Growth very friable and bleeds very readily on touching. Apparently some thickening in left utero-sacral ligament per rectum. The parts seemed freely movable.

*Diagnosis.*—Squamous-epithelioma of cervix.

*Operation.*—Vaginal hysterectomy, the posterior vaginal fornix being also removed, revealing the peritoneum of Douglas' pouch free from growth. Clamps and ligatures.

*Parts removed* showed the whole cervix to be distended and invaded by growth, the cervix being as large as the fundus. There was a thin shell of cervical tissue everywhere on the growth, which thus seemed to have been quite removed.

*Progress of the case* was uneventful. Recovery.

*After-history.*—Recurred. Died November, 1900.

CASE 95.—H. R., æt. 29. Dr. Galabin, Queen 19, May 26th, 1899. Married, four children, the last two years ago. Five months before admission a brownish discharge was noted between the monthly periods. The discharge continued, the periods remained normal in duration, amount and interval. There was no pain. No micturition troubles.

*On examination.*—The cervix was enlarged, irregular, with nodules of stony hardness. A deep laceration on each side allowed the finger to enter the canal and caused bleeding. Mobility was impaired.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. No difficulties. Clamps and ligatures used. Both ovaries were removed.

*Parts removed* showed that the growth extended up to the internal os, and also that the line of incision on the left side passed through the growth where it had extended into the cellular tissue outside the cervix.

*Progress of the case.*—Clamps removed in thirty-six hours. Later progress uneventful. Recovery.

*After-history.*—May 10th, 1901. Reports herself quite well.

CASE 96.—F. S., æt. 34. Dr. Galabin, Queen , October 7th, 1899. Married, four children, one miscarriage. The last pregnancy being ten months previously. This last pregnancy terminated at the sixth month and had been accompanied by irregular hæmorrhages all the time. Three months after this premature birth she menstruated and continued losing more or less ever since (seven months before admission). Latterly she had pain, but not enough to keep her awake at night.

*On examination.*—An irregular warty growth of the cervix which bled readily was found. It extended on to the vaginal wall on the left side. The whole cervix seemed much broadened. There was considerable fixation of the uterus.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy, no difficulty, but one clamp slipped, and the hæmorrhage had to be arrested by several pairs of pressure forceps, which were left on. Ligatures for the upper halves of the broad ligaments.

*Parts removed* showed that the whole cervix was involved in growth, and the body of the uterus was just reached. On each side the growth reached the cut surface of the cervix.

*Progress of the case* was uneventful. Recovery.

*After-history.*—Recurrence took place and death occurred in October, 1900.

CASE 97. H. B., æt., 58. Dr. Galabin, Queen 2, December 20th, 1898. Married, one child, thirty-three years previously. Admitted for hæmorrhage and pain. The menopause seemed to have occurred at fifty, but there had been many irregular hæmorrhages since that time. A year before her admission she had a constant blood-stained discharge attended with some abdominal pain.

*On examination.*—The cervix was atrophied and the uterine body not enlarged, quite movable and slightly retroverted. Scrapings from the endometrium were taken and proved microscopically to be columnar-celled carcinoma.

*Operation.* Vaginal hysterectomy. A perineal incision was first made on account of the small vulval orifice. Clamps and ligatures were used for the broad ligaments.

*Parts removed* showed a somewhat irregular uterine cavity, owing to the presence of some atrophic fibro-myomata. The whole lining of the uterus was involved by the growth, which nowhere extended deeply into the muscular substance. The growth had just reached the cervical canal.

*Progress of the case* was uneventful. Clamps thirty-six hours. Ligatures came away on the fourteenth day. Recovery.

*After-history.*—May 10th, 1901. Reports herself as quite well and free from her former complaint.

CASE 98.—J. A., æt. 60. Dr. Horrocks, Queen 2, November 7th, 1899. Married, four children, the last nineteen years previously. Admitted for hæmorrhage, the menopause having occurred eleven years previously. The hæmorrhage came on six months before admission and was first noticed on defæcation. There had been no pain at any time and no difficulty with micturition.

*On examination.*—A rounded mass one and a half inches in diameter was found in the region of the os uteri. It extended over to the left fornix, was hard, not friable but bled readily on examination.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy, complicated somewhat by the extensive involvement of the left broad ligament. Hæmostasis by twenty-four pairs of Spencer Wells' forceps.

*Parts removed* showed that the whole cervix was involved in growth and there was some doubt if the incision of the left broad ligament was outside the margin of the growth.

*Progress of the case* was uneventful, except for some incontinence of urine which gradually got better. Apparently there was no fistula. Recovery.

*After-history.*—The growth recurred and the patient died in November, 1900.

CASE 99.—J. G., æt. 52. Dr. Horrocks, Queen 9, June 5th, 1899. Married, five children, the last eleven years previously. Menopause at 48. Present illness began six months before admission with a hæmorrhage, followed by a profuse discharge. The hæmorrhage continued on and off and the discharge became offensive. Pain in lower part of abdomen, back, and thighs. No micturition troubles.

*On examination.*—The cervix could be felt much enlarged by growth, especially in front. The surface of the growth was irregular, nodular and ulcerated. It was a question whether the bladder wall was infiltrated or not. Bleeds readily on examination. Under an anæsthetic some thickening in the right broad ligament was felt.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. Some difficulty in separating the bladder. Spencer Wells' forceps used for hæmostasis of the broad ligaments.

*Parts removed* showed the whole cervix and part of the body of the uterus involved in growth. On the right side a nodule of growth had been cut through. No pus or blood in cavity of uterus.

*Progress of the case.*—There was some hæmorrhage for three days after the operation, and then a period of foul discharge for several days, otherwise uneventful. Recovery.

*After-history.*—May, 1901. Reports herself as being now as bad as ever after having been fairly well for two months after the operation.

CASE 100.—J. B., æt. 62. Dr. Horrocks, Queen , June 8th, 1899. Married, four children, the last nineteen years previously. Admitted for growth of cervix. Menopause nine years previously (52). Seven months before admission noticed a watery blood-stained discharge, which was very offensive at times. No serious hæmorrhage. Difficulty of micturition for five or six months. No mention of pain in the report. Had had some prolapse of the uterus at one time.

*On examination.*—A lobulated cauliflower growth of anterior lip of cervix.

*Diagnosis.*—Epithelioma of cervix.

*Operation.*—Vaginal hysterectomy. No difficulty. Hæmostasis by fifteen pairs of Spencer Wells' forceps.

*Parts removed* showed that the growth had only slightly infiltrated the anterior lip of the cervix. The supra-vaginal cervix was elongated, and the body small.

*Progress of the case.*—Uneventful. Recovery.

*After-history.*—Cannot be traced.

CASE 101.—S. P., æt. 60. Dr. Horrocks, Queen 2, June 15th, 1899. Married, ten children; the last twenty-one years previously. Menopause at 50 years. Had had a foul discharge for four and a half months, which had been blood-stained for three weeks. No micturition trouble. No pain.

*On examination.*—The cervix was found enlarged, hard and warty. Slight involvement of left fornix by the growth. Uterus freely movable. Slight bleeding on touching growth.

*Diagnosis.*—Carcinoma of cervix.

*Operation.*—Vaginal hysterectomy. No difficulty. Twenty-one pairs of Spencer Wells' forceps applied to broad ligaments.

*Parts removed* showed that the whole cervix was involved in the growth, but was not much enlarged by it. Body of the uterus dilated somewhat, and contained a little foul pus. Walls of uterus a little thickened.

*Progress* uneventful. Recovery.

*After-history.*—May 10th, 1901. Is reported to be in perfect health.

CASE 102.—S. M., æt. 60. Dr. Horrocks, Queen 2, June 21st, 1899. Married, two children; the last twenty-five years previously. Admitted for hæmorrhage. Menopause seven years previously. Had a hæmorrhage five months before admission, which, at first excessive, soon lessened, but became continuous. There had been no pain. No micturition troubles.

*On examination.*—Uterine body found uniformly enlarged, sound passed three and a half inches. Soft to the touch as if containing fluid or soft growth. Small fragments removed with curette for examination. Free bleeding after this examination. Microscopically, the growth was a carcinoma.

*Operation.*—Abdominal pan-hysterectomy. The broad ligaments were ligatured, and the vagina closed by sutures. No great difficulty, but a little hæmorrhage at the base of the broad ligaments gave some trouble.

*The parts removed* showed that the growth involved the whole body of the uterus, except a small area near the fundus about half an inch in diameter. The growth was about one and a half inches thick, and approached to within one-quarter inch of the peritoneum. Cervix unaffected.

*Progress of the case* was uneventful. Recovery.

*After-history.*—Cannot be traced.



**LIST**  
 OF  
**GENTLEMEN EDUCATED AT GUY'S HOSPITAL**  
 WHO HAVE PASSED THE  
 EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,  
 &c., &c.,  
 IN THE YEAR 1898.

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**University of Oxford.**

*Final Examination for the Degrees of Bachelor of Medicine and Surgery.*

F. E. Fremantle.		R. O. Moon.
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**University of Cambridge.**

*Degree of Doctor of Medicine.*

A. V. Clarke.		M. Craig.
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*Final Examination for the Medical and Surgical Degrees.*

Part I.

S. E. Denyer.		H. A. Gaitskell.		F. J. Nicholls.
A. C. Fry.		F. A. Godson.		F. Shufflebotham.
		W. K. Wills.		

Part II.

A. B. Carter.		S. E. Denyer.		F. J. Lidderdale.
		W. K. Wills.		

*Second Examination for the Medical and Surgical Degrees.*

Part I.

A. G. Harvey.

Part II.

J. A. Andrews.		E. Bigg.		H. Wachter.
H. Bentley.		F. Richmond.		J. A. Wood.

*First Examination for the Medical and Surgical Degrees.*

**Part II.**

H. A. Cutler.

*Examination in Sanitary Science.*

**Parts I. and II.**

O. Beven.	W. H. Jewell.	R. W. C. Pierce.
W. E. Dixon.	A. E. Larking.	S. Worthington.
O. Eaton.	W. J. Lubeck.	

**University of London.**

*Examination for the Degree of Doctor of Medicine.*

A. P. Allan.	W. H. Jewell.	E. Ivens Spriggs.
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*Examination for the Degree of Master in Surgery.*

C. H. Fagge.

*Examination for the Degree of Bachelor of Surgery.*

**First Division.**

D. J. Munro.

*Obtained Honours.*

**Second Division.**

H. W. Bruce.	L. E. C. Handson.	W. H. M. Telling.
E. Coleman.	R. H. J. Swan.	

*Examination for the Degree of Bachelor of Medicine.*

**May.**

**First Division.**

A. J. Cleveland.

**Second Division.**

W. N. East.	R. T. Fitz-Hugh.	R. Kay.
	D. L. Smith.	

**October.**

**Second Division.**

R. H. J. Swan.

*Obtained First-class Honours in Medicine.*

V. E. Collins.

*Obtained Honours in Obstetric Medicine and First-class Honours in Forensic Medicine.*

D. J. Munro.	A. G. G. Plumley.
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*Obtained Honours in Obstetric Medicine.*

T. P. Berry.	L. E. C. Handson.	P. W. Moore.
J. A. Glover.	J. Howell.	W. H. M. Telling.

*Intermediate Examination in Medicine.*

January.

*Excluding Physiology.*

Second Division.

E. T. Jensen.	J. T. M. McDougall.	D. L. Morgan.
	A. Pearson.	

*Physiology only.*

Second Division.

P. T. Manson.

July.

Honours Examination.

S. Hodgson.

*Obtained Honours in Physiology and Histology.*

Entire Examination.

Second Division.

W. R. Dunstan.	J. A. B. Hammond.	K. V. Trubshaw.
F. G. Gibson.	C. Tessier.	

*Excluding Physiology.*

Second Division.

R. M. Barron.	D. Forsyth.	A. C. Ransford.
F. A. Beattie.	T. A. Matthews.	M. J. Rees.
J. D. Bridger.	S. J. Ormond.	

*Physiology only.*

Second Division.

A. Fraser.	J. T. M. McDougall.	A. Moon.
	D. L. Morgan.	

*Preliminary Scientific (M.B.) Examination.*

January.

Entire Examination.

First Division.

H. C. C. Mann.

Second Division.

E. W. Strange.		H. Tipping.
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*Chemistry and Experimental Physics.*

H. S. Brown.

*Biology.*

K. Anderson.	J. Braithwaite.	L. G. Nash.
A. J. Beadel.	A. W. Iredell.	

**July.**

**Entire Examination.**

**First Division.**

W. F. Box.		F. Rogerson.		H. D. Smart.
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**Second Division.**

F. W. Fawcett.		C. E. Iredell.		A. H. Snell.
J. J. Gardiner.		C. D. Pye-Smith.		A. M. Webber.
		B. H. Wedd.		

*Chemistry and Experimental Physics.*

P. A. S. Dyson.		P. A. Peall.
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*Biology.*

H. S. Brown.		P. W. Hamond.
--------------	--	---------------

*Intermediate Examination in Science and Preliminary Scientific Examination conjointly.*

*Examination for Honours.*

P. R. Bolus.

*Obtained First-class Honours in Inorganic Chemistry.*

E. H. B. Milsom.

*Obtained Honours in Inorganic Chemistry.*

*B.Sc. Examination.*

**Second Division.**

A. E. H. Pakes.

**University of Durham.**

*Examination for the Degree of Doctor of Medicine.*

G. Gocher.		H. M. Meyrick-Jones.		T. B. Poole.
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*Examination for the Degree of Doctor of Medicine for Practitioners of Fifteen Years' Standing.*

W. L. Chubb.		A. W. Emms.		L. W. K. Phillips.
W. Darley-Hartley.		J. M. McCarthy.		

*Examination for Degree of Master in Surgery.*

J. W. H. Eyre.

*Final Examination for the Degrees of Bachelor of Medicine and Surgery.*

G. Burton-Brown.		A. M. Rygate.		J. Harris.
		T. J. A. Tulk-Hart.		

**Royal College of Physicians of London.**

*Admitted to the Fellowship.*

H. J. Campbell.

*Final Examination for the License.*

January.

S. E. Denyer.	F. W. Lee.	H. D. Peile.
J. E. Dupigny.	W. J. Lindsay.	G. H. Steele.
W. A. Fuller.	P. W. Moore.	J. W. Sweetlove.
J. A. Glover.	G. N. Mottram.	A. H. Turner.
W. A. Horton.	C. R. Nicholson.	A. J. Wernet.

April.

E. Ashby.	E. S. Hall.	Ed. Evans.
H. Bernard-Onraët.	M. C. Hayward.	A. H. Godson.
S. C. Clapham.	F. J. Lidderdale.	E. D. Hancock.

July.

H. P. W. Barrow.	L. Humphry.	J. C. S. Peatson.
H. W. Graham.	W. J. S. Harvey.	A. A. Price.
E. G. Goddard.	S. H. Mason.	W. C. Pritchard.
L. E. C. Handson.	W. C. Milward.	C. Shepherd.

October.

K. B. Alexander.	F. E. Hutchinson.	D. Rice.
R. Balderston.	L. H. McGavin.	W. S. Richardson.
T. P. Berry.	F. J. H. Martin.	L. T. A. Rowland.
H. L. Eason.	A. W. Nourse.	A. H. M. Seward.
H. E. C. Fox.	G. C. Owsley.	H. Simson.
J. Harris.	S. B. Pennington.	W. H. M. Telling.
F. H. R. Heath.	J. E. Powell.	

**Royal College of Surgeons of England.**

*Final Examination for the Fellowship.*

H. T. S. Bell.	A. H. Godson.	A. W. Ormond.
	W. I. Hancock.	

*First Examination for the Fellowship.*

H. B. Bailey.	F. G. Cross.	H. McD. Parrott.
P. W. L. Camps.	H. Davies-Colley.	J. F. Robinson.
B. Copley.	D. G. Greenfield.	N. F. Ticehurst.

*Final Examination for the Membership.*

**January.**

S. E. Denyer.	F. W. Lee.	H. D. Peile.
J. E. Dupigny.	W. J. Lindsay.	G. H. Steele.
W. A. Fuller.	P. W. Moore.	J. W. Sweetlove.
J. A. Glover.	G. N. Mottram.	A. H. Turner.
W. A. Horton.	C. R. Nicholson.	A. J. Wernet.

**April.**

E. Ashby.	E. S. Hall.	Edw. Evans.
H. Bernard-Onraët.	M. C. Hayward.	A. H. Godson.
S. C. Clapham.	F. J. Lidderdale.	E. D. Hancock.

**July.**

H. P. W. Barrow.	L. Humphry.	J. C. S. Peatson.
E. G. Goddard.	W. J. S. Harvey.	A. A. Price.
H. W. Graham.	S. H. Mason.	W. C. Pritchard.
L. E. C. Handson.	W. C. Milward.	C. Shepherd.

**October.**

K. B. Alexander.	F. E. Hutchinson.	D. Rice.
R. Balderston.	L. H. McGavin.	W. S. Richardson.
T. P. Berry.	J. J. H. Martin.	L. T. A. Rowland.
H. L. Eason.	A. W. Nourse.	A. H. M. Seward.
H. E. C. Fox.	G. C. Owsley.	H. Simson.
J. Harris.	S. B. Pennington.	W. H. M. Telling.
F. H. R. Heath.	J. E. Powell.	

**Royal College of Surgeons of Edinburgh.**

*Examination for the Fellowship.*

C. G. F. Morice.

**Society of Apothecaries of London,**

*Final Examination for the License.*

J. C. Bell.	H. W. Graham.	M. Jenkins.
E. Fryer.	W. J. Henson.	H. Munro.

## MEDALLISTS AND PRIZEMEN.

JULY, 1899.

### *Open Scholarships in Arts.*

Robert Fenton Macdonald, Bancroft School, Woodford Wells, £100.  
George Hamilton, Aske's School, Hatcham, £50.

### *Dental Students.*

John Walker Walton, Folkestone Grammar School, £30.

### *Open Scholarships in Science.*

Edwin Henry Britton Milsom, Christchurch, New Zealand, £150.  
Frederick Rogerson, Guy's Hospital, £30 } equal.  
Neville Ivens Spriggs (Private Study), £30 }  
Herbert Douglas Smart, Guy's Hospital, Certificate.  
Henry Swarrick Brown, Guy's Hospital, Certificate.

### *Scholarship for University Students.*

Herbert Stanley French, Christ Church, Oxford, £50.  
Ercoyde Ihler Claxton, King's College, Cambridge, Certificate.

### *Junior Proficiency Prizes.*

Carrick Hey Robertson, £20.  
Joseph Tremayne Hicks, £15.  
Albert Ernest Hardman Pakes, £5. } equal.  
Alfred Herbert Edwin Wall, £5. }  
Percival Pasley Cole, Certificate.

### *The Michael Harris Prize for Anatomy.*

Carrick Hey Robertson, Prize, £10.  
Alfred Herbert Edwin Wall, Certificate.  
William Gibson Parker, Certificate.  
Harold Thornbury Palmer, Certificate.

### *The Wooldridge Memorial Prize for Physiology.*

Joseph Tremayne Hicks, £5  
Albert Ernest Hardman Pakes, £5 } equal.  
Percival Pasley Cole, Certificate.  
Ellis Gordon Goldie, Certificate.

### *The Hilton Prize for Dissection (1898).*

Claude Tessier, £5.

### *The Arthur Durham Prizes for Dissections.*

#### *First Year's Students.*

Alexander Moxen Webber, Prize, £5.  
Neville Ivens Spriggs, Certificate.  
Charles Derwent Pye-Smith, Certificate.

*Senior Students.*

Carrick Hey Robertson, £7 10s.  
 Alfred Herbert Edwin Wall, £7 10s. } equal.

*Dental Prizes.**Second Year's Students (1898).*

Percival Sidney Campkin, Prize, £15.  
 John Stewart Farnfield } equal Certificates.  
 Ernest Noel Mason }

*First Year's Students.*

Charles Sculthorpe Morris, Prize, £10.  
 John Bennett, Certificate.

*Practical Dentistry Prize.*

Charles Sculthorpe Morris, Prize, £10.  
 John Bennett, Certificate.

*Senior Proficiency Prizes.*

Caleb Thomas Hilton, £20.  
 Ernest Blair Dowsett, £15.  
 George Norman Meachen, £10.  
 Alfred Reid, Certificate.

*The Richard Bredin Prize for Clinical Study.*

Russell Henry Jocelyn Swan, £25.

*The Golding-Bird Gold Medal and Scholarship for Sanitary Science.*

Stanley Copley, £20.  
 Claude Martin Vernon, Certificate.  
 Harry James Spon, Certificate.

*The Treasurer's Gold Medal for Clinical Medicine.*

Graham Scales Simpson.

*The Treasurer's Gold Medal for Clinical Surgery.*

Caleb Thomas Hilton.



## THE PHYSICAL SOCIETY.

Honorary President.—SIR SAMUEL WILKES, Bart., M.D., LL.D., F.R.S.

## Presidents.

H. W. Bruce, M.B., A. H. Davies, B.A., F. E. Fremantle, M.A., M.B., B.Ch., C. T. Hilton, A. H. B. Kirkman, G. N. Meachen, W. T. Milton, M.B., B.S., G. E. Richmond, B.A., M.B., B.S., B.Sc., F. Shufflebotham, B.A., F. O. Støhr, B.A., R. H. J. Swan, F. D. Turner, P. Turner, B.Sc., F. E. Walker.

Honorary Secretaries.—J. H. Targett, M.S., G. Bellingham Smith, B.S.

## PRIZEMEN FOR THE SESSION 1898-9.

The First and Second Prizes were divided equally between Mr. H. W. Bruce, for his paper on "Plague," and Mr. F. E. Walker, for his paper on "Burns and Scalds."

The Treasurer's Prize was awarded to Mr. W. T. Milton, for "Pancreatic Inflammation and Hæmorrhage."

## CLINICAL APPOINTMENTS HELD DURING THE YEAR 1898.

## HOUSE PHYSICIANS.

H. P. Ferraby.	W. E. Hills.	G. K. Levick.
A. E. Porter.	R. W. Mayston.	A. J. Cleveland.
H. J. Starling.	W. N. East.	

## HOUSE SURGEONS.

A. W. Ormond.	R. B. Stamford.	M. H. Way.
F. S. Batchelor.	P. Turner.	G. E. Richmond.
C. R. Hodgson.	C. W. Booker.	

## ASSISTANT HOUSE SURGEONS.

H. Leader.	M. H. Thornely.	F. E. Fremantle.
F. A. Godson.	S. E. Denyer.	Edward Evans.
C. Jephcott.		

## RESIDENT OBSTETRIC ASSISTANTS.

R. W. Mayston.	J. N. Gardiner.	C. W. Booker.
C. R. Hodgson.	R. T. Fitz-Hugh.	E. I. Spriggs.
W. T. Milton.	F. A. Godson.	C. P. Weekes.
F. S. Batchelor.	A. E. Porter.	V. E. Collins.

## CLINICAL ASSISTANTS.

C. R. Hodgson.	M. H. Thornely.	W. T. Milton.
G. E. Richmond.	P. Turner.	F. E. Fremantle.
F. W. Moore.	A. H. B. Kirkman.	E. G. Goddard.
H. M. Berncastle.	Edward Evans.	H. Cardin.
J. M. F. Brickdale.	H. W. Bruce.	J. A. Glover.
H. D. Peile.	W. J. Lindsay.	F. Shufflebotham.
K. B. Alexander.	A. H. Carter.	H. E. C. Fox.
G. C. Owsley.	F. E. Walker.	E. W. S. Rowland.

## GYNÆCOLOGICAL ASSISTANTS.

C. P. Weekes.	P. W. Moore.	J. A. Glover.
R. T. Fitz-Hugh.	W. T. Milton.	E. I. Spriggs.
W. Mussellwhite.	S. E. Denyer.	F. E. Fremantle.
H. J. Starling.	F. W. Brook.	F. E. Walker.
A. H. B. Kirkman.	L. E. C. Handson.	T. P. Berry.
L. H. McGavin.		

## CLINICAL ASSISTANTS IN THE MEDICAL WARDS.

W. S. Richardson.	A. H. M. Saward.	S. C. Clapham.
T. W. H. Landon.	W. H. M. Telling.	A. B. Passmore.
E. W. S. Rowland.	K. R. Alexander.	A. A. Miller.
L. H. McGavin.	R. H. J. Swan.	H. L. Eason.
A. R. Thomas.	W. L. Baker.	F. D. Turner.
W. R. Cazenove.		

## CLINICAL ASSISTANTS IN THE SURGICAL WARDS.

L. D. B. Cogan.	C. P. Weekes.	H. Leader.
F. A. Godson.	T. M. Walker.	T. P. Berry.
F. W. Brook.	H. J. Starling.	C. B. Sells.
W. R. Cazenove.	J. G. Taylor.	C. D. Outred.
R. W. B. Hall.	H. Durbridge.	A. A. Smith.
L. Wilkin.		

## SURGEONS' DRESSERS.

S. A. Ruzzak.	E. E. Parrett.	H. Durbridge.
W. L. Baker.	A. Reid.	J. J. Rodil.
A. E. Clarke.	O. Marriott.	C. F. Watson.
D. J. Munro.	F. W. Brook.	F. Shuffelebotham.
H. L. Eason.	L. H. McGavin.	W. R. Cazenove.
J. G. Taylor.	L. Wilkin.	H. A. Gaitskell.
A. H. Carter.	R. H. J. Swan.	F. D. Turner.
W. B. Hope.	A. R. MacLachlan.	J. T. Dunston.
E. J. Tongue.	H. S. Dismorr.	C. A. Lower.
C. B. Sells.	G. T. Brundrett.	E. I. Davis.
A. A. Smith.	H. C. Holden.	G. Shorland.
J. B. Brockwell.	C. H. Brangwin.	R. H. Denny.
C. Edwards.	F. O. Stoeher.	W. H. Edwards.
J. H. Roberts.	G. M. Brown.	H. M. Reeve.
G. N. Meachen.	F. E. Hutchinson.	L. C. Martin.
A. W. Penrose.	T. W. S. Browne.	C. B. Thomson.
F. R. Featherstone.	E. W. Goble.	T. E. Holman.
P. H. Ward.	C. E. Hicks.	B. W. Moss.
O. T. Hilton.	G. S. Simpson.	W. G. Stewart.
D. P. Watson.	W. B. Secretan.	W. M. Thomas.
R. W. B. Hall.	H. M. Hardy.	M. A. Alabone.
P. C. P. Ingram.	A. E. Cawston.	A. H. Davies.
H. Braund.	H. St. A. Alder.	C. A. D. Bryan.
T. Morgan.	A. W. Talbot.	J. N. Dyson.
J. F. Northcott.	R. C. Mullins.	R. M. Barron.
T. J. Wright.	C. H. Glenn.	E. Stott.
A. Densham.		

## ASSISTANT SURGEONS' DRESSERS.

J. H. Roberts.	G. M. Brown.	E. G. Andrew.
J. B. C. Brockwell.	E. I. Davis.	G. N. Meachen.
G. Shorland.	E. A. Evans.	V. T. C. Bent.
T. W. S. Browne.	H. R. H. Denny.	H. M. Reeve.
C. A. D. Bryan.	E. W. H. Shenton.	E. M. Judge.
A. A. Smith.	H. B. Foster.	C. Edwards.
W. H. Edwards.	W. McIlroy.	H. C. Holden.
A. W. Penrose.	C. T. Hilton.	G. S. Simpson.
R. W. B. Hall.	H. M. Hardy.	L. C. Martin.
J. T. M. McDougall.	E. W. Goble.	W. Johnson.
W. M. Thomas.	T. E. Holman.	M. A. Alabone.
P. H. Ward.	F. J. Felix Jones.	E. P. Mitchell.
C. E. Hicks.	B. W. Moss.	W. B. Secretan.
W. G. Stewart.	D. P. Watson.	T. J. Wright.
J. F. Northcott.	R. C. Mullins.	M. A. Alabone.
E. H. Cragg.	A. W. Talbot.	C. H. Glenn.
A. E. Cawston.	H. Braund.	H. St. A. Alder.
R. M. Barron.	E. Stott.	A. H. Davies.
J. T. M. McDougall.	E. Cohen.	W. H. Brailey.
A. M. Dallas.	T. E. Holmes.	A. C. Osburn.
A. C. Lewis.	H. V. Bagshawe.	E. A. Miller.
T. P. Thomas.	H. B. Carr.	P. D. Hunter.
H. A. Higgins.	E. H. Felton.	J. Atkins.
A. Fraser.	J. C. J. Da Silva.	G. Lewin.
J. S. S. Perkins.		

## DENTAL SURGEONS' DRESSERS.

A. H. B. Kirkman.	J. H. Jones.	R. Fell.
B. Isaac.	F. E. Fremantle.	S. C. Clapham.
H. M. Berncastle.	E. G. Goddard.	F. H. R. Heath.
J. Harris.	J. E. Powell.	T. W. S. Browne.

## CLINICAL ASSISTANTS IN MEDICAL OUT-PATIENTS.

E. S. Hall.	W. C. Pritchard.	S. B. Pennington.
C. C. Worts.	J. E. Powell.	M. A. Alabone.
S. A. Ruzzak.	W. L. Baker.	A. R. Thomas.
C. H. Brangwin.	J. T. Dunston.	

## DRESSERS IN THE EYE WARDS.

W. N. East.	W. C. Milward.	A. Kinsey-Morgan.
H. P. W. Barrow.	A. D. Lewis.	W. Mussellwhite.
A. B. Passmore.	J. Harris.	L. E. C. Handson.
A. H. M. Seward.	W. S. Richardson.	C. Shepherd.
W. H. M. Smith.	E. S. Hall.	F. G. Thomas.
S. B. Pennington.	W. C. Pritchard.	H. L. Eason.
T. M. Walker.	J. T. Dunston.	W. B. Hope.
J. H. Jones.	H. C. Sturdy.	J. Howell.
G. Beley.	H. Simson.	A. R. McLachlan.
W. R. Cazenove.	E. A. Longhurst.	

## CLERKS IN THE EYE WARDS.

T. W. S. Browne.	R. Balderstone.	W. H. Edwards.
J. E. H. Parsons.	N. F. Ticehurst.	A. G. Osborn.
F. E. Walker.	W. L. Baker.	J. G. Taylor.
L. H. McGavin.	H. N. Clark.	F. Curtis.
H. C. Sturdy.	A. E. Clarke.	A. B. Passmore.
R. H. J. Swan.	C. H. Brangwin.	E. J. Tongue.
E. W. S. Rowland.	J. F. Robinson.	C. D. Outred.
E. R. Row.	A. A. Smith.	F. L. Rae.
L. Wilkin.	H. Simson.	H. M. Berncastle.
C. F. Watson.	G. T. Wrench.	H. D. Peile.
W. M. Thomas.	W. R. Cazenove.	J. E. Powell.
F. R. Featherstone.	O. Marriott.	A. A. Miller.
V. J. Crawford.	W. B. Hope.	E. G. Goddard.
S. A. Ruzzak.	A. R. Thomas.	L. Humphry.
H. L. Eason.	J. H. Jones.	R. J. Pritchard.

## DRESSERS IN THE THROAT DEPARTMENT.

J. M. F. Brickdale.	J. Howell.	A. J. Cleveland.
E. G. Goddard.	J. Howells.	S. E. Denyer.
H. M. Berncastle.	W. J. Lindsay.	W. E. Hills.
R. Balderstone.	F. J. Nicholls.	H. D. Peile.
H. Thornely.	F. E. Walker.	A. H. Carter.
A. B. Passmore.	T. P. Berry.	G. C. Owsley.
J. L. Payne.	E. R. Row.	A. W. Ormond.
H. Durbridge.	L. H. McGavin.	W. H. M. Telling.
E. B. Dowsett.	F. Shufflebotham.	

## CLERKS IN THE THROAT DEPARTMENT.

E. R. Row.	H. P. W. Barrow.	E. B. Dowsett.
W. H. M. Telling.	A. Reid.	H. M. Reeve.

## MEDICAL WARD CLERKS.

L. C. Martin.	T. B. Fawley.	A. W. Penrose.
E. W. Goble.	C. B. Sells.	C. T. Hilton.
W. B. Secretan.	C. E. Hicks.	W. G. Stewart.
R. W. B. Hall.	G. S. Simpson.	F. O. Stoehr.
D. P. Watson.	H. M. Hardy.	E. Stott.
C. B. Thomson.	D. M. Evans.	R. M. Barron.
J. Atkins.	A. Fraser.	T. J. Wright.
H. St. A. Alder.	A. Densham.	J. S. Cousins.
A. H. Davies.	A. E. Cawston.	J. F. Northcott.
E. H. Felton.	F. R. Featherstone.	R. C. Mullins.
A. W. Talbot.	C. H. Glenn.	H. A. Higgins.
A. M. Thomas.	H. Braund.	W. H. Brailey.
E. T. Jensen.	B. W. Moss.	J. A. Glover.
G. Lewin.	E. Cohen.	H. V. Bagshawe.
H. B. Carr.	E. A. Miller.	P. D. Hunter.
A. C. Lewis.	J. C. J. Da Silva.	J. S. S. Perkins.
T. P. Thomas.	T. E. Holmes.	E. M. Judge.
H. B. Foster.	T. T. Kelly.	D. G. Greenfield.
F. J. F. Jones.	F. A. Segreda.	R. D. Atwood.
J. E. L. Bates.	T. R. Beale Brown.	F. G. Cross.
W. P. Ker.	H. Mc D. Parrott.	E. Shelton Jones.
L. E. Stamm.	R. Tilbury.	J. L. Whatley.
J. M. Brydone.	E. J. F. Hardenberg.	E. G. Andrew.
A. D. E. Kennard.	A. Moon.	J. D. Bridger.
W. Johnson.	W. H. Loosely.	E. P. Mitchell.
H. E. Morris.	B. Muir.	A. C. Ransford.
J. A. Butler.	P. W. L. Camps.	D. Forsyth.
P. S. Mandy.	A. Pearson.	L. Pern.
H. G. Rashleigh.	E. F. Reeve.	G. G. Gibson.
K. W. Goadby.	R. S. Roper.	F. D. Welch.
G. T. Willan.	H. Bentley.	J. A. Wood.

## EXTERN OBSTETRIC ATTENDANTS.

A. E. Clarke.	S. A. Ruzzak.	D. J. Munro.
F. W. Brook.	J. F. Dunston.	F. Shufflebotham.
W. H. M. Telling.	F. E. Walker.	J. L. Payne.
G. H. Bedford.	E. W. S. Rowland.	E. A. Longhurst.
R. Michell.	E. R. Row.	H. C. Sturdy.
E. B. Dowsett.	G. T. Brundrett.	T. P. Berry.
H. S. Crapper.	V. T. C. Bent.	A. A. Miller.
J. T. De Coteau.	W. H. Edwards.	A. H. Carter.
G. C. Owsley.	H. E. C. Fox.	H. Durbridge.
W. B. Hope.	W. McIlroy.	T. H. Body.
E. W. H. Shenton.	F. R. Featherstone.	H. L. Eason.
W. R. Cazenove.	J. G. Taylor.	F. D. Turner.
J. Matthews.	C. B. Thomson.	C. H. Brangwin.
E. J. Tongue.	A. R. McLachlan.	A. Reid.
T. M. Walker.	A. A. Smith.	E. E. Parrett.
J. H. Roberts.	L. Wilkin.	P. C. P. Ingram.
H. M. Reeve.	H. A. Gaitskell.	A. C. Ambrose.
R. M. Barron.	R. O. Jones.	A. G. Osborn.
H. R. H. Denny.	J. T. M. McDougall.	C. J. Francis.
O. Marriott.	H. B. Dismorr.	C. Edwards.
H. M. Hardy.	G. N. Meachen.	T. E. Holman.
C. E. Hicks.	J. B. C. Brockwell.	F. O. Stoehr.

## SURGICAL WARD CLERKS.

P. D. Hunter.	W. Johnson.	A. C. Lewis.
H. V. Bagshawe.	H. B. Carr.	F. J. F. Jones.
E. A. Miller.	E. P. Mitchell.	J. C. J. Da Silva.
G. Lewin.	W. H. Brailey.	J. A. Glover.
E. T. Jensen.	J. M. Brydone.	T. E. Holmes.
J. S. S. Perkins.	T. T. Kelly.	H. E. Morris.
R. D. Attwood.	T. R. Beale-Brown.	W. P. Ker.
P. T. Manson.	E. Shelton Jones.	R. Tilbury.
J. L. Whatley.	M. D. Wood.	E. J. F. Hardenberg.
H. Davies-Colley.	A. Moon.	L. E. Stamm.
J. E. L. Bates.	F. A. Segrada.	W. H. Loosely.
J. A. Butler.	P. W. L. Camps.	F. Curtis.
D. Forsyth.	P. S. Mandy.	B. Muir.
A. Pearson.	H. G. Rashleigh.	E. F. Reeve.
J. Evans.	K. W. Goadby.	G. S. C. Hayes.
A. C. Osburn.	L. Pern.	S. L. Prall.
R. S. Roper.	F. D. Welch.	G. T. Willan.
T. B. Fawley.	A. D. Kennard.	H. Bentley.
J. A. Wood.	K. V. Trubshaw.	N. F. Ticehurst.
J. E. Collins.	S. Hodgson.	H. K. Lacey.
T. A. Matthews.	S. J. Ormond.	F. A. Beattie.
J. A. B. Hammond.	L. Hirsch.	R. Jiménez.
H. D. Kempthorne.	R. P. Marshall.	D. L. Morgan.
P. J. Nash.	F. M. N. Ommanney.	S. S. H. Shannon.
F. W. Smith.	H. Wachter.	V. M. Wallis.
F. E. Welchman.	D. W. Smith.	G. S. Graham-Smith.
E. G. Wales.	F. W. Sime.	E. C. Bevers.
T. H. B. Dobson.	G. Clarke.	

## ASSISTANT SURGEONS' CLERKS.

N. F. Ticehurst.	A. H. Davies.	M. D. Wood.
J. F. Robinson.	B. Rodil.	F. G. Gibson.
J. M. F. Brickdale.	R. J. Pritchard.	D. L. Morgan.
F. D. Turner	A. Fraser.	T. Morgan.
E. G. Andrew.	J. E. Powell.	H. P. W. Barrow.
T. H. W. Landon.	H. Davies-Colley.	P. T. Manson.
E. A. Miller.	H. Wachter.	L. Hirsch.
F. Curtis.	G. G. Davidson.	M. J. Rees.
H. S. French.	E. I. Claxton.	L. E. Stamm.
R. D. Attwood.	C. R. Howard.	

## AURAL SURGEONS' DRESSERS.

J. E. Dupigny.	L. Humphry.	A. D. Passmore.
R. J. Pritchard.	W. H. M. Telling.	F. E. Walker.
E. W. S. Rowland.	A. H. B. Kirkman.	H. J. M. Barker.
T. P. Berry.	J. L. Payne.	J. Matthews.
J. N. Gardiner.	S. A. Ruzzak.	J. F. Robinson.
H. E. C. Fox.	R. Balderstone.	R. Dowsett.
V. T. C. Bent.	H. Durbridge.	A. W. Ormond.
A. A. Miller.	E. R. Row.	T. W. S. Browne.

## OBSTETRIC WARD CLERKS.

H. J. Starling.	H. R. Miller.	J. Howells.
E. A. Longhurst.	H. C. Sturdy.	A. M. Dallas.
G. Shorland.		

## OBSTETRIC OUT-PATIENTS' CLERKS.

G. H. Bedford.	H. E. Dismorr.	T. H. W. Landon.
S. E. Denyer.	C. D. Outred.	V. J. Crawford.
H. W. Fox.	M. A. Alabone.	B. B. Gough.
C. H. Brangwin.	J. T. M. McDougall.	E. J. Tongue.
R. Michell.	H. C. Sturdy.	D. Rice.
W. H. M. Telling.	R. T. De Coteau.	O. Marriott.
R. Balderstone.	F. Shufflebotham.	T. P. Berry.
W. B. Hope.	A. M. Dallas.	A. G. Osborn.
W. H. Edwards.	W. R. Cazenove.	J. Matthews.
J. G. Taylor.	L. Wilkin.	A. R. McLachlan.
E. M. Judge.		

## ASSISTANT PHYSICIANS' CLERKS.

A. G. C. Davies.	J. T. M. McDougall.	P. H. Ward.
W. B. Secretan.	T. E. Holman.	M. A. Alabone.
T. Morgan.	W. M. Thomas.	E. H. Cragg.
A. W. Fowell.	R. W. B. Hall.	C. H. Glenn.
A. M. Thomas.	A. W. Talbot.	W. W. Harrison.
A. Fraser.	H. Braund.	H. B. Carr.
T. E. Holmes.	H. A. Higgins.	J. S. S. Perkins.
A. M. Dallas.	A. Densham.	E. M. Judge.
F. A. Segreda.	E. Shelton-Jones.	D. G. Greenfield.
T. T. Kelly.	R. D. Attwood.	J. L. Whatley.
T. R. B. Browne.	P. Ker.	F. J. F. Jones.
J. E. L. Bates.		

CLERKS IN THE SKIN DEPARTMENT.

V. J. Crawford.	S. B. Pennington.	G. S. Simpson.
J. E. Powell.	F. G. Gibson.	E. Stott.
R. H. J. Swan.	A. M. Dallas.	A. Reid.
H. M. Reeve.	H. A. Gaitskell.	

POST-MORTEM CLERKS.

L. E. C. Handson.	C. D. Outred.	T. P. Berry.
H. P. W. Barrow.	P. H. Ward.	F. E. Walker.
J. Matthews.	J. E. Powell.	E. W. S. Rowland.
A. R. Thomas.	H. N. Clark.	D. Rice.
R. H. J. Swan.	F. D. Turner.	L. H. McGavin.
W. L. Baker.	A. G. Osborn.	D. J. Munro.
C. F. Watson.	H. Durbridge.	E. J. Tongue.
E. E. Parrett.	J. H. Roberts.	S. A. Ruzzak.
C. B. Thomson.	H. A. Gaitskell.	A. W. Penrose.
J. G. Taylor.	A. Reid.	G. N. Meachen.

DENTAL SURGEONS DRESSERS.

A. H. B. Kirkman.	J. H. Jones.	R. Fell.
J. Harris.	J. E. Powell.	T. W. S. Browne.
H. M. Berncastle.	E. G. Goddard.	F. H. R. Heath.
B. Isaac	F. E. Fremantle.	S. C. Clapham.

CLERKS IN THE ELECTRICAL DEPARTMENT.

E. J. Tongue.	J. M. Brydone.	T. E. Holmes.
J. D. Bridger.	F. O. Stoechr.	

CLERKS TO ANÆSTHETISTS.

C. Shepherd.	J. Howells.	T. M. Walker.
K. B. Alexander.	A. R. McLachlan.	H. P. W. Barrow.
W. B. Hope.	T. E. Holman.	F. E. Walker.
L. Wilkin.	E. H. Cragg.	C. T. Hilton.
F. E. Saffery.	E. B. Dowsett.	W. M. Thomas.
C. A. Lower.	P. W. Moore.	T. P. Berry.
E. J. Tongue.	T. H. Pody.	J. T. M. McDougall.
A. W. Powell.	J. Matthews.	L. D. B. Cogan.
A. A. Miller.	R. T. Fitz-Hugh.	V. J. Crawford.
A. C. Ambrose.	L. Humphry.	G. M. Brown.
E. R. Row.	L. H. McGavin.	G. H. Bedford.
E. I. Davis.	J. M. Brydone.	E. W. S. Rowland.
W. S. Richardson.	J. G. Taylor.	A. Armer.
R. H. J. Swan.	W. R. Cazenove.	W. P. Grellet.
R. Kay.	J. T. Dunston.	F. G. Cross.
S. H. Longhurst.	H. E. C. Fox.	D. J. Munro.
A. H. Carter.	C. F. Watson.	J. E. Powell.
A. W. Talbot.	A. E. Clarke.	A. W. Nourse.
E. H. Felton.	H. Durbridge.	H. L. Eason.
W. H. Edwards.	J. N. Dyson.	H. Simson.
E. D. Hancock.	C. B. Sells.	P. C. P. Ingram.
R. C. Mullins.	J. T. De Coteau.	P. H. Ward.
F. J. Nicholl.	C. C. Worts.	R. W. B. Hall.
E. E. Parrett.	T. H. W. Landon.	J. E. H. Parsons.
G. T. Brundrett.		

**DENTAL SCHOOL.****APPOINTMENTS HELD DURING THE YEAR 1898.****DENTAL HOUSE-SURGEONS.**

G. O. Betts.	J. K. Clark.	S. J. Redpath.
E. B. Dowsett.	D. P. Tracy.	R. Umney.

**ASSISTANT DENTAL HOUSE-SURGEONS.**

P. S. Campkin.	T. H. Wilkinson.	E. N. Mason.
C. H. Huckle.	S. H. Olver.	J. S. Farnfield.
L. M. Fleetwood.	F. Warlow.	

**DEMONSTRATORS IN THE CONSERVATION ROOM.**

J. S. Farnfield.	E. N. Mason.	T. F. Ryan.
L. M. Fleetwood.	H. W. Morris.	S. H. Olver.
P. H. H. Palmer.	E. R. Howlett.	F. Warlow.
H. T. Campkin.	S. H. Jones.	A. E. Rowlett.

**ASSISTANT DEMONSTRATOR OF DENTAL MICROSCOPY.**

L. M. Fleetwood.

**DRESSERS IN THE EXTRACTION ROOM.**

A. E. Rowlett.	S. J. St. H. Tweney.	R. W. Allen.
E. B. L. White.	W. H. Loosely.	E. R. Howlett.
A. M. Gabriel.	J. H. Wilkes.	H. T. Campkin.
F. Warlow.	J. Black.	R. C. G. May.
S. L. Pallant.	J. A. Whittington.	J. S. Biss.
S. H. Longhurst.	P. H. H. Palmer.	R. J. Morrell.
S. H. Jones.	W. W. C. Jones.	P. P. Cole.
H. L. Shelton.	G. H. Drake.	J. B. Morrish.
A. C. Edwards.	C. H. Bubb.	R. Peacock.
G. P. Pollitt.	L. Webb.	E. Couchman.
W. Jarvis.	F. J. Pearce.	A. H. Saunders.
A. E. Wood.	R. B. Recordon.	F. R. Coish.
W. H. Tattersfield.	P. Greenwood.	J. H. Greenwood.
H. Hatton.	C. F. Rose.	A. Battersby.
N. P. Shepherd.	H. Maurice.	F. W. Garman.
H. R. Shapland.		

**DRESSERS IN THE GAS ROOM.**

H. W. Morris.	J. Black.	J. S. Farnfield.
J. G. McAlpin.	T. F. Ryan.	L. C. A. Knight.
W. H. Loosely.	A. J. Gwatkin.	A. E. Rowlett.
D. P. Tracy.	P. E. Chandler.	A. L. Lambert.
E. N. Mason.	J. R. S. Ash.	M. P. Nathan.
P. H. H. Palmer.	T. E. Norton.	P. S. Campkin.
L. M. Fleetwood.	W. H. Phillips.	H. H. Evans.
C. F. Witcomb.	S. H. Longhurst.	W. P. Crombie.
E. B. L. White.	E. R. Howlett.	W. C. C. Jones.
S. H. Jones.	J. S. Biss.	L. Webb.
E. G. Walton.	J. A. Whittington.	G. L. Lewis.
W. Jarvis.	F. Warlow.	F. J. Pearce.
H. H. Evans.	R. B. Recordon.	C. F. Rose.
J. H. Wilkes.	B. F. Henry.	A. Battersby.
H. T. Campkin.	A. M. Gabriel.	J. B. Morrish.
H. Maurice.	F. R. Coish.	



JUNIOR DRESSERS IN THE CONSERVATION ROOM.

H. T. Campkin.	A. M. Gabriel.	J. H. Wilkes.
R. O. G. May.	R. J. Morrell.	F. J. Pearce.
G. P. Pollitt.	G. H. Drake.	R. Peacock.
R. B. Recordon.	W. H. Tattersfield.	F. W. Garman.
A. H. Saunders.	N. P. Shepherd.	A. E. Wood.
F. R. Coish.	J. H. Greenwood.	C. F. Rose.
H. Hatton.	H. R. Shapland.	P. Greenwood.
J. L. Wartski.	C. S. Morris.	W. R. Searle.
C. R. Shattock.	G. H. Steweni.	A. Archer.
A. Drewitt.	A. M. A. Stevens.	A. W. Aldis.
J. Bennett.	H. R. C. Butler.	G. H. Aylen.
A. R. Cummings.	W. A. Dennant.	P. H. Furnivall.
H. N. Hillier.	E. G. Smith.	

DRESSERS IN THE CONSERVATION ROOM.

J. R. S. Ash.	H. H. Evans.	A. Hughes.
E. R. Howlett.	A. L. Lambert.	F. Warlow.
C. F. Witcomb.	A. C. Carpenter.	H. Charnock.
A. J. Gwatkin.	C. F. Jessop.	G. J. Lewis.
W. H. Loosely.	E. G. Walton.	A. A. Bartholomew.
W. W. C. Jones.	L. C. A. Knight.	J. G. McAlpin.
E. B. Marshall-Frost.	T. E. Norton.	W. H. Phillips.
P. E. Chandler.	S. Cock.	R. J. Morrell.
M. P. Nathan.	A. E. Rowlett.	F. W. S. Stone.
R. Umney.	J. Harper.	C. H. Huckle.
W. Jarvis.	F. J. Pearce.	A. H. Saunders.
F. P. Uttley.	E. C. Bartlett.	J. S. Biss.
S. H. Jones.	R. P. Millett.	P. H. H. Palmer.
D. P. Tracy.	J. A. Whittington.	S. H. Longhurst.
F. R. Coish.	G. H. Drake.	F. W. Garman.
J. H. Greenwood.	H. Maurice.	E. B. L. White.
H. Knight.	H. L. Shelton.	P. P. Cole.
R. B. Recordon.	G. P. Pollitt.	C. E. Rose.
A. E. Wood.	R. C. G. May.	R. Peacock.
S. L. Pallant.	W. H. Tattersfield.	J. Black.
E. Couchman.	N. P. Shepherd.	R. W. Allen.
H. T. Campkin.	S. J. St. H. Tweney.	A. M. Gabriel.
H. Hatton.	H. R. Shapland.	J. H. Wilkes.
A. C. Edwards.	C. H. Bubb.	L. Webb.
W. P. Crombie.	L. M. Fleetwood.	S. H. Olver.
J. B. Morrish.	C. F. Jessop.	A. Battersby.
W. A. Dennant.	S. L. Prall.	P. H. Furnivall.
A. Drewitt.	C. S. Morris.	C. R. Shattock.
H. C. Winckworth.	A. R. Cummings.	H. N. Hillier.
E. G. Smith.	G. H. Steweni.	A. Archer.
H. R. C. Butler.	J. Bennett.	W. R. Searle.
K. C. Ness.	W. Jarvis.	A. W. Aldis.
G. H. Aylen.	P. Greenwood.	J. L. Wartski.
M. N. Mitchener.		

# GUY'S HOSPITAL.

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## MEDICAL AND SURGICAL STAFF. 1901.

**Consulting Physicians.**—SIR SAMUEL WILKS, BART., M.D., LL.D.,  
F.R.S.; F. W. PAVY, M.D., LL.D., F.R.S.; P. H. PYE-SMITH,  
M.D., F.R.S.; J. F. GOODHART, M.D., LL.D.

**Consulting Surgeons.**—J. BIRKETT, Esq.; THOMAS BRYANT, M.Ch.

**Consulting Obstetric Physician.**—H. OLDHAM, M.D.

**Physicians & Assistant Physicians. Surgeons & Assistant Surgeons.**

FREDERICK TAYLOR, M.D.

W. HALE WHITE, M.D.

G. NEWTON PITT, M.D.

E. C. PERRY, M.D.

L. E. SHAW, M.D.

J. W. WASHBOURN, M.D.

J. H. BRYANT, M.D.

J. FAWCETT, M.D.

H. G. HOWSE, M.S.

R. CLEMENT LUCAS, B.S.

C. H. GOLDING-BIRD, M.B.

W. H. A. JACOBSON, M.Ch.

CHARTERS J. SYMONDS, M.S.

W. ARBUTHNOT LANE, M.S.

L. A. DUNN, M.S.

A. D. FRIPP, M.S.

F. J. STEWARD, M.S.

**Obstetric Physicians.**

A. L. GALABIN, M.D.

P. HORROCKS, M.D.

**Assistant Obstetric Physician.**

J. H. TARGETT, M.S.

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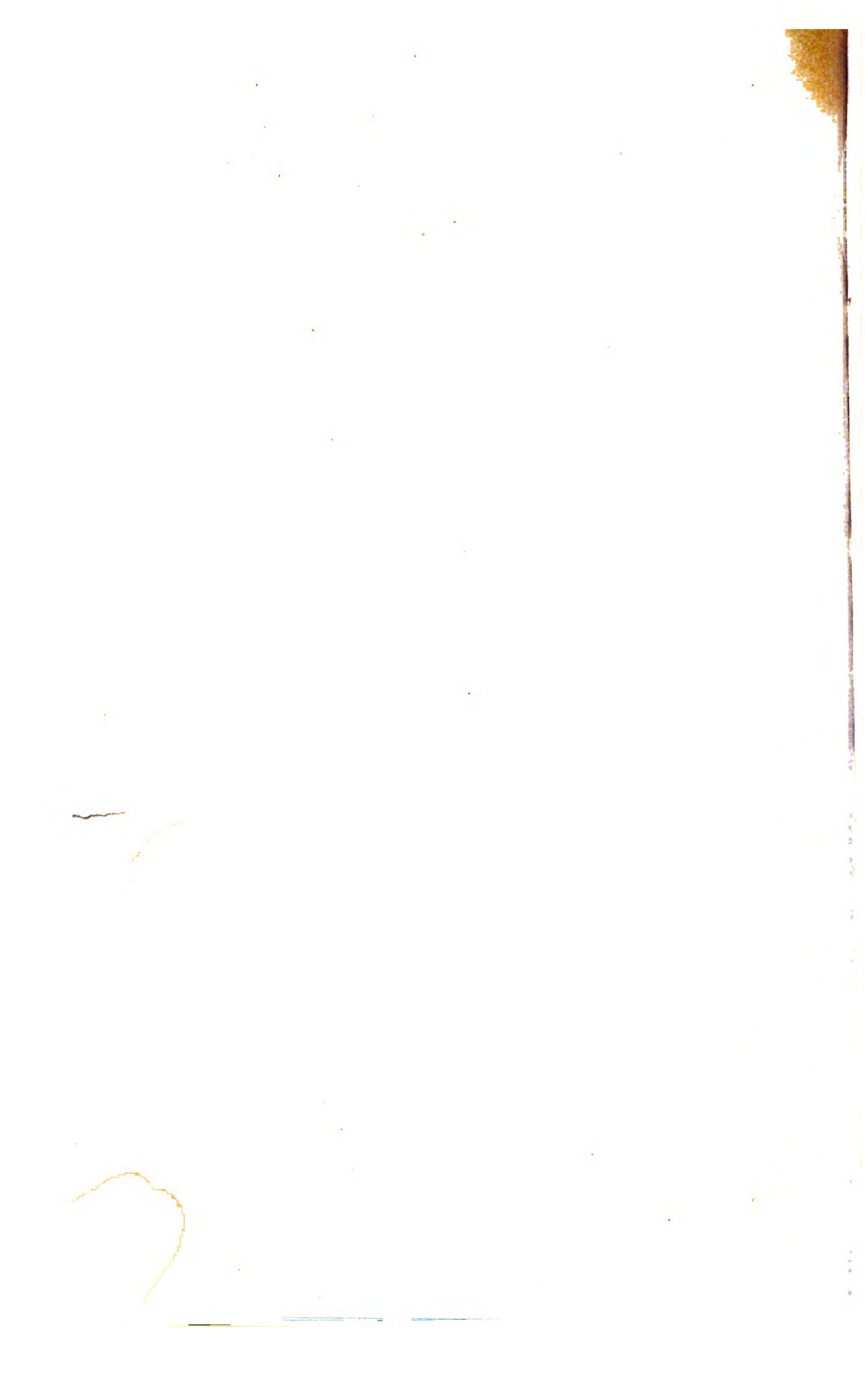
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Dental Appointments held during the year 1897.

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